This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

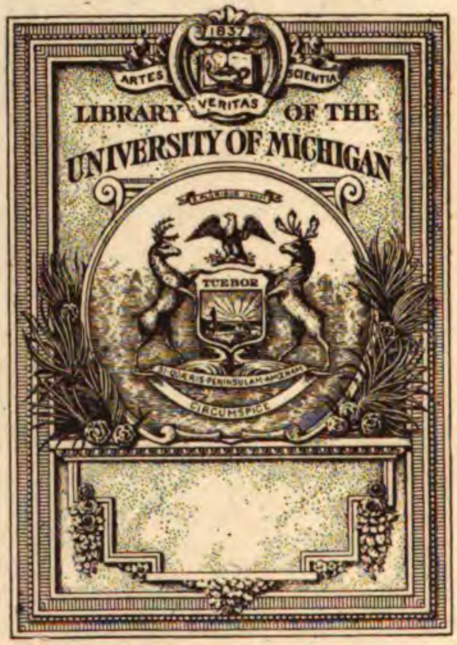
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

B 3 9015 00205 438 8
University of Michigan – BUHR


LIBRARY OF THE UNIVERSITY OF MICHIGAN

610.5
A67
D5

THE
ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

FOUNDED AND EDITED BY
HEINRICH STERN, MD., LL.D.
NEW YORK

VOLUME IX
1916

PUBLISHED BY
REBMAN COMPANY
141, 143 AND 145 WEST 36TH STREET
NEW YORK, N.Y.

1916

COPYRIGHT, 1916, BY
REBMAN COMPANY

All Rights Reserved

THE

ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX JANUARY, 1916 No. 1

FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York

PUBLISHED BY
REBMAN COMPANY
141, 143 AND 145 WEST 36TH STREET
NEW YORK, N.Y.

COPYRIGHT 1916 BY REBMAN COMPANY. ALL RIGHTS RESERVED.

Entered as Second-Class Matter, February 8, 1908, at the Post-Office at New York, N. Y.
Under the Act of Congress of March 3, 1879.

SUBSCRIPTION TWO DOLLARS A YEAR. SINGLE COPIES 75 CENTS. FOREIGN $2.50.

VOLUME IX

Contributors of Special Articles

Ager, Louis C.
(Brooklyn-New York)
Barach, Joseph H.
(Pittsburgh, Pa.)
Basch, Seymour
(New York)
Cornwall, Edward E.
(Brooklyn-New York)
Deaver, John B.
(Philadelphia, Pa.)
Dennett, Roger Herbert
(New York)
Epstein, J.
(New York)
Epstein, Sigmund
(New York)
Fowler, Russell S.
(Brooklyn-New York)
Friedman, G. A.
(New York)
Gallant, A. Ernest
(New York)
Gordon, Alfred
(Philadelphia, Pa.)
Hart, T. Stuart
(New York)
Hays, Harold
(New York)
Lehnhoff, Henry J.
(Lincoln, Neb.)
Lerch, Otto
(New Orleans)
Levy, I. H.
(Syracuse, N. Y.)
Levy, I. J.
(New York)
Lichty, John A.
(Pittsburgh, Pa.)
Nascher, I. L.
(New York)
Nettle, Armin
(New York)
Neuhof, Harold
(New York)
Reilly, Thomas F.
(New York)
Sheffield, Herman B.
(New York)
Stein, Herbert E.
(New York)
Sturmdorf, Arnold
(New York)
Vander Hoof, Douglas
(Richmond, Va.)
Weinberger, William
(New York)
Williams, B. G. R.
(Paris, Ill.)

329813

Plates

FACING PAGE

Round Cell Sarcoma (Ponto-Cerebellar Tumor) 68
Echinococcus Cyst of Left Lobe of Liver 213
Abdominal Disease of Orthopedic Origin 328

Illustrations in Text

PAGE

Orthodiagram of Normal Heart 3
Diagram Showing Unavoidable Error in Percussing Cardiac Outlines 5
Diagram Showing Cardiac Dilatation and Hypertrophy 6
Diagram, Parenchymatous Myocarditis 6
Orthodiagramatic Measurements 7
Orthodiagrams (2 Illustrations) 9
Orthodiagrams (2 Illustrations) 10
Diagram Illustrating Percussion Methods, etc. (3 Illustrations) 11
The Same (2 Illustrations) 12
The Same 13
The Same (2 Illustrations) 14
The Same 15
The Same (2 Illustrations) 16
The Same (2 Illustrations) 17
The Same 18
The Same (2 Illustrations) 19
The Same 20
The Same 21
Abnormalities of Myocardial Function 35
Abnormalities of Myocardial Function 36
Abnormalities of Myocardial Function (5 Illustrations) 42
Abnormalities of Myocardial Function 43
Abnormalities of Myocardial Function (2 Illustrations) 44
Abnormalities of Myocardial Function (2 Illustrations) 45
Abnormalities of Myocardial Function (2 Illustrations) 47
Abnormalities of Myocardial Function 48
Abnormalities of Myocardial Function 50
Abnormalities of Myocardial Function 51
Abnormalities of Myocardial Function (3 Illustrations) 53
Abnormalities of Myocardial Function (3 Illustrations) 54
Abnormalities of Myocardial Function (2 Illustrations) 56
Abnormalities of Myocardial Function 59
Abnormalities of Myocardial Function (2 Illustrations) 62
Gouty Etiology in Joint Affections (3 Illustrations) 201
Diagram Showing Relation of Endocardial Murmurs to Regular Heart Sounds 262

INDEX OF SUBJECTS

Page reference in bold-face type indicates a Special Article or General Retrospect; in ordinary type, an Original Abstract of an Article that has appeared in full elsewhere.

PAGE
Abdominal diseases, the diagnosis of some, of orthopedic origin **327**
Abortion reaction in children, epidemic 76
Abscess, parapharyngeal 77
Acetone body production in children 377
Acid-fast bacilli and joint disease 73
Acidity and pepsin digestion in the stomach of the nursling.. 186
Acidity determination of gastric juice 171
Acidity determination of the filtrated and unfiltrated gastric juice, quantitative 171
Acidity of the nursling's stomach 80
Acidosis in children.......... 72
Acidosis occurring with diarrhea 155
Acidosis of children, the relation of migraine to the so-called.. **230**
Acidosis of chronic nephritis.... 155
Addison's syndrome in old people 376
Adenitis, tonsils and chronic cervical 76
Adhesions in the cecal region, bands and **108**
Agglutination tests in inoculated persons 163
Albumin determinatian in gastric contents, Salomon's 173
Albumin in urine, detection of.. 157
Albuminometer, source of error with Esbach's 72
Albuminuria, renal efficiency determination in nephritis and orthotic 181
Alexia 85
Amino-acid minimum for maintenance and growth.......... 277
Aneurism of hepatic artery...... 384
Aneurism of the thoracic aorta.. 283
Antirabic treatment, paralysis during 282
Antitrypsin reaction in clinical diagnosis 375
Aorta, aneurism of the thoracic 283
Aorta, syphilis and.......... 169

PAGE
Aortitis, luetic, clinical diagnosis of **295**
Aortitis, syphilitic 170
Appendicectomies, ineffective.... 386
Appendicitis as sequela of tonsillitis 83
Appendicitis, Bastedo's sign in chronic 386
Appendicitis, etiology of.......... 174
Appendicitis, pseudo 387
Appendix vermiformis, extraperitoneal 83
Artery, hepatic, aneurism of.... 384
Aspermatism 288
Asthma, chronic bronchitis, emphysema and **126**
Atherosclerosis, adolescent and beginning 79
Baby, the normal.......... 396
Bacilli, acid-fast, and joint disease 73
Bacilli, systemic infection and dysentery 162
Bacillus abortus 381
Bacteriology of peritoneal exudate 84
Bacteriology of septic wounds.. 75
Band, Lane's kink and.......... **116**
Bands and adhesions in the cecal region **108**
Basedow's disease, effect of iodthyroglobulin and thyronucleid on the nitrogen metabolism and the blood-picture of myxedema and 375
Beta-oxybutyric acid in urine of normal children, ketones and.. 154
Blood in gastric contents and feces, occult 173
Blood pressure, determinations. 78
Blood pressure, increased, and physical blood changes....... 375
Blood serum and urine, amylase in the 276
Blood smears, oxydase reaction on 156
Blood stain, a new.......... 155
Blood sugar content, effects of surgical procedures on........ 277

PAGE
Blood, test for demonstrating large increase of leukocytes in the 375
Bock's stethoscope 384
Bordet-Gengou bacillus, complement-fixation reactions of.... 74
Brain sclerosis, a new familial form of diffuse..... 390
Breast, tuberculosis of..... 280
Bronchial glands 77
Bronchitis, emphysema and asthma, chronic 126
Cachexia of pituitary origin.... 159
Calcium content of bile and the formation of biliary calculi... 83
Calculi calcium content of bile and the formation of biliary.. 83
Cancer of the stomach and the Röntgen rays 316
Cannabis indica, increase of the effect of narcotics by..... 378
Carbohydrate metabolism in hyperthyroidism 158
Carcinoma, gastric 174
Carcinoma of the vulva, primary 394
Cardiac diseases, the diagnosis of . 79
Carmin test in infants and children 72
Cases, diagnostic errors in senile 130
Cecal region, bands and adhesions in the 108
Cerebral intoxication, meningism and meningitis, clinical distinctions between 85
Cerebrospinal fluid, functions of 379
Cerebrospinal fluid in paresis, colloidal gold precipitating substance in 180
Cerebrospinal fluid, reducing substances in 389
Cerebrospinal liquid in syphilis, infectivity of 181
Cerebrospinal meningitis, etiology of epidemic 380
Child, effects of nutrition of mother on 292
Childhood, d'Espine's sign in.... 166
Childhood, Röntgen-ray in lobar pneumonia in 74
Children, acetone body production in 377
Children, acidosis in..... 72
Children, acute non-epidemic inflammation of the sublingual glands in 79

PAGE
Children, carmin test in infants and 72
Children, chronic retention of urine in 181
Children, diarrhea and vomiting in 285
Children, epidemic abortion reaction in 76
Children, fainting attacks in.... 285
Children, intestinal parasites in.. 177
Children, non-protein nitrogenous constituents of blood and phenolsulphonephthalein test in 277
Children, palpation of peripheral glands 157
Children, the relation of migraine to the so-called acidosis of... 230
Children, thymus disease in..... 312
Cholecystitis catarrhalis subacuta, an early type of gallbladder disease 214
Cholelithiasis 175
Chorea 389
Chorea, etiology of..... 281
Chvostek's sign 389
Colons, morphography of..... 177
Complement-fixation in tuberculosis 383
Complement-fixation reactions of Bordet-Gengou bacillus 74
Complement-fixation test, gonorrheal 289
Cord symptoms, fracture of the vertebrae without 179
Cyst of the left lobe of the liver discharging into the left hepatic duct, echinococcus.... 209
Dermatitis exfoliativa neonatorum 186
D'Espine sign, diagnostic significance of 77
D'Espine's sign in childhood.... 166
Diabetes and war..... 377
Diabetes innocuus 376
Diabetes in surgical diseases, glycosuria and 157
Diabetes, latent syphilis and.... 280
Diabetes mellitus, mortality from 377
Diarrhea, acidosis occurring with 155
Diarrhea and vomiting of children 285
Diarrhea in infants, infectios... 282
Diarrhea of infants, bacteriology of feces in..... 388

PAGE

Diarrheas in bottle-fed infants, differential diagnosis of...... 143
Diazo and urochromogen reactions as prognostic aids in pulmonary tuberculosis 279
Diazo reaction, permanganate reaction and 164
Digestion, delayed gastric...... 284
Digestion-lipemia 378
Digestive tract, radiology of the 27
Diphtheria bacilli, guinea-pig test for the virulence of.......... 162
Diphtheria in the first year of life 280
Drop percussion, comparison of, with the methods now in use and orthodiography 1
Ducts, the etiology and diagnosis of infective diseases of the biliary ducts 100
Duodenal ulcer and the Röntgen rays 316
Duodenal ulcer, diagnosis of.... 173
Duodenal ulcer, indications for operative interference in...... 91
Duodenal ulcers, end results of operation on perforated gastric and 172
Duodenal ulcers, gastric and.... 81
Duodenum, carcinoma of the suprapapillary 386
Dysentery bacilli, systemic infection and 162
Dysentery due to unknown causes 178
Dysentery, observations on..... 388
Echinococcus cyst of the left lobe of the liver discharging into the left hepatic duct............ 209
Edema, Pathogenesis of Quincke's 159
Edema, pulmonary, the prognosis of 120
Emphysema, asthma and bronchitis 126
Endamebiasis tonsilar, and thyroid disturbances 73
Energometric observations in the pregnant, parturient and lying-in woman 88
Enteritis complicating measles.. 162
Enteroptosis, diagnostic methods in Glenard's disease.......... 267
Epilepsy in offspring of epileptics 389
Epileptic attack, death as the consequence of the........... 86
Epileptics, bacillus isolated from 390

PAGE

Esbach's albuminometer, source of error with................ 72
Exophthalmic goiter bruit over the eye ball in................ 158
Extrauterine Pregnancy, a symptom in 139
Extremities, puerperal gangrene of the 379
Exudate, bacteriology of peritoneal 84
Exudates, peritoneal 84
Eye findings in pulmonary tuberculosis 166
Fainting attacks in children..... 285
Fertility and sterility........... 392
Fever, an initial sign of tuberculosis in infants............. 165
Fever, transitory, of the newborn 185
Gall-bladder disease, cholecystitis catarrhalis subacuta, an early type of 214
Gall-bladder, gastric chemism in affections of the............. 174
Gall-stone disease 175
Gall-stones, accumulation of pus or bile beneath the diaphragm as a consequence of the presence of 387
Gangrene, puerperal, of the extremities 379
Gangrenous wounds, bacteria of 75
Gas and gas infections, Röntgen-ray diagnosis of............. 282
Gastric and duodenal ulcers.... 81
Gastric and duodenal ulcers, end results of operation on perforated 172
Gastric chemism in affections of the gall-bladder 174
Gastric digestion, delayed....... 284
Gastric secretion, examination of normal, by the fractional method 284
Gastric ulcer, Röntgen rays in the diagnosis of 316
Gastroenteric tract of infants, permeability of, to undigested protein 285
Genital system, tuberculosis of the male 87
Gland, clinical observations on the pineal 278
Gland, experimental observations on the pineal............ 278
Glands, bronchial 77

PAGE

Glands in children, acute non-epidemic inflammation of the sublingual 79
Glands, peripheral, palpation of, in children 157
Glenard's disease, diagnostic methods in 267
Glioma of the eye with papid recurrence after enucleation.... 69
Gluzinski's method in the diagnosis of gastric disease....... 171
Glycosuria and diabetes in surgical diseases 157
Goiter, blood-picture in, and its alteration after operation..... 376
Goiter, bruit over the eye ball in exophthalmic 158
Gonorrheal complement-fixation test 289
Gonorrhea in women, diagnosis of 185
Gout, pulmonary affections and.. 78
Growth, amino-acid minimum for maintenance and 277
Growth and sexual development, influence of pituitary feeding upon 159
Gruber-Widal reaction 381
Gunshot wounds of the solid abdominal viscera 286
Gynecological urology 395
Heart and kidneys, syphilis with secondary involvement of..... 168
Heart block, rheumatic.......... 170
Heart, clinical diagnostics of diseases of the..............233, 331
Heart disease and pregnancy.... 89
Heart disease, congenital, some varieties of 385
Heart diseases in pregnancy, estimation of the various forms of 185
Heart, slight enlargement of..... 170
Heart, soldier's 170
Heart sound, third.............. 386
Heart's action in jaundice...... 171
Hemoglobin, influence of age and sex on 379
Hemolytic jaundice 381
Hemoptysis due to high tension 79
Hemorrhages, occult, in ulcer and carcinoma of the stomach..... 173
Hepatic disease, excretion of certain urinary constituents in.... 176

PAGE

Hydrops intermittens, familial, and purin metabolism......... 377
Hyperindicanemia in persons with and without kidney disease, indicanemia and......... 154
Hypernephroma of the kidney, the origin of................ 287
Hyperthyroidism, carbohydrate metabolism in 158
Ileus and movable kidney, intermittent 83
Indicanemia and hyperindicanemia in persons with and without kidney disease............ 154
Infancy, prognosis of pyelosystitis in 288
Infancy, Röntgen-ray examination in tuberculosis of........ 74
Infants and children, carmin test in 72
Infants, bacterial affections of the urinary organs in......... 186
Infants, bacteriology of feces in diarrhea of 388
Infants, differential diagnosis of diarrheas in bottle-fed........ 143
Infants, infectious diarrhea in.. 282
Infants, fever an initial sign of tuberculosis in 165
Infants, permeability of gastroenteric tract of, to undigested protein 285
Infants, technic for intravenous injections in 395
Infants, vaginitis in............ 395
Infective diseases of the biliary ducts, the etiology and diagnosis of 100
Intraabdominal disease, the value of deep percussion in the diagnosis of subacute............. 98
Internal secretions, myopathy and disorders of 278
Intestine, activity of physostigmin and pilocargin combinations upon the surviving...... 388
Jaundice, heart's action in....... 171
Jaundice, hemolytic 381
Joint affections, on the recognition of the gouty etiology in chronic 191
Joint disease, acid-fast bacilli and 73
Ketones and Beta-hydrooxybutyric acid in urine of normal children 154

PAGE
Kidney, disease, indicanemia and hyperindicanemia in persons with and without............. 154
Kidney. intermittent ileus and movable 83
Kidney, the origin of hypernephroma of the............. 287
Kink and band, Lane's.......... 116
Kiutsi-Malone urinary test for pregnancy 185
Labor, contracted pelvis and difficult 90
Labor, retraction of uterine muscle and obstructed............ 393
Lane's kink and band........... 116
Leukemia during pregnancy, a case of 89
Leukocytes in malignant disease 156
Leukocytes in the blood, test for the demonstration of a large increase of 375
Leukocytes, neutrophile, in malaria and yellow fever........ 74
Lipase and lymphocytes......... 157
Lipemia, digestion 378
Lipodystrophia progressiva 377
Liver function in normal pregnancy, a study of............. 290
Lobar pneumonia in childhood, Röntgen-ray in 74
Lues, reinfection in............. 382
Luetic aortitis, clinical diagnosis of 295
Luetin reaction in congenital syphilis 168
Lymph glands, bacteria associated with abnormal 163
Lymphatics, tuberculosis of the cevical 383
Lymphocytes, lipase and......... 157
Malaria and yellow fever, neutrophile leukocytes in......... 74
Malaria, diagnosis of........... 161
Malignancy, the clinical and laboratory manifestations of.. 302
Malignant disease, leukocytes in 1
Mammary gland, tuberculosis of 166
Measles. enteritis complicating.. 162
Meningitis as determined by laboratory examination 160
Meningitis, clinical and bacteriological observations in epidemic 159
Meningitis, clinical distinction between cerebral intoxication, meningism and 85

PAGE
Meningitis, etiology of epidemic cerebrospinal 380
Meningitis in the newborn and in infants under three months of age 281
Meningococci in nasopharynx... 74
Menstruation and psychosis..... 86
Menstruation, vicarious 393
Metabolism in hyperthyroidism, carbohydrate 158
Mesenteric vessels in abdominal disease, superior 172
Migraine and ocular defects..... 84
Migraine, the relation of, to the so-called acidosis of children.. 230
Mind, disorders of, relation of pelvic diseases to............. 179
Mucormycosis of the stomach... 386
Myocardial efficiency, Bocks stethoscope in determining....... 384
Myocardial function, the diagnosis of abnormalities of...... 31
Myopathy and disorders of internal secretions 278
Myxedema and cretinism, the effect of some thyroid preparations on the metabolism and the blood-picture of.......... 376
Narcotics, increase of the effect of, by cannabis indica........ 378
Nasopharynx. meningococci in.. 74
Nephritis, acute, in war participants 391
Nephritis, acute syphilitic....... 169
Nephritis and orthotic albuminuria, renal efficiency determination in 181
Nephritis, hemorrhagic, in war participants 391
Nephritis, acidosis of........... 155
Nephritis, termination of acute.. 391
Nephritis, the early diagnosis and treatment of chronic.......... 218
Nephritis, vascular versus interstitial 227
Nerve, sensory field of facial.... 178
Nervous system, effect of high explosives on the central...... 178
Neuritis and neuralgia, periostitis and osteitis as a cause of local pains and so-called........... 180
Newborn and infants under three months of age, meningitis in.. 281
Newborn infants, pyloric hypertrophy in 81

PAGE
Newborn, transitory fever of the 185
Ninhydrin color reactions of proteins and their split products.. 276
Nitrogen in urine, determination of 378
Nonne and Froin's spinal fluid syndromes 287
Nursling, acidity and pepsin digestion in the stomach of the.. 186
Nursling, renal secretion in the 395
Nursling's stomach, acidity of the 80
Nutrition of mother, effects of, on child 292
Occiput posterior, a statistical study of 635 labors with the.. 291
Ocular defects, migraine and.... 84
Opsiuria 87
Orthodiography, comparison of drop percussion with the methods now in use and.......... 1
Osteogenesis imperfecta 278
Ovaries, metastatic carcinoma of 184
Oxydase reaction on blood smears 156
Pains, local, and so-called neuritis and neuralgia, periostitis and osteitis as a cause of......... 180
Palpatory percussion, two varieties of 22
Pancreatic secretion 82
Pancreatic secretion, methods of testing the external........... 177
Paralysis during Pasteur antirabic treatment 282
Paralysis, early diagnosis of acute infantile 205
Parapharyngeal abscess 77
Parasites in children, intestinal. 177
Paratyphoid fever, fatal cases of 163
Paresis, colloidal gold precipitating substance in cerebrospinal fluid in................. 180
Pelvis, contracted, and difficult labor 90
Pelvis, relation of diseases of the, to mental disorders........... 179
Pepsin and trypsin in the fasting stomach, detection of......... 80
Percussion, drop 1
Percussion, the value of deep, in the diagnosis of subacute intraabdominal disease 98
Percussion, two varieties of palpatory 22
Pericarditis 384

PAGE
Peritoneal exudates 84
Permanganate (urochromogen) reaction and Diazo reaction... 164
Phenolsulphonephthalein test in children 277
Pineal gland, clinical observations on the 277
Pineal gland, experimental observations on the............ 278
Pituitary feeding, influence of upon growth and sexual development 159
Pituitary origin, cachexia of.... 159
Pneumococcal throat 281
Pneumonia, cold air and blood pressure in 383
Pneumothorax, spontaneous 169
Poliomyelitis, early diagnosis of acute 205
Ponto-cerebellar tumors, diagnostic elements of............ 31
Pregnancy, a case of leukemia during 89
Pregnancy, a study of liver function in normal............... 290
Pregnancy, a symptom in extrauterine 139
Pregnancy, estimation of the various forms of heart diseases in 185
Pregnancy, functional determination of renal activity during.. 89
Pregnancy, genuine typhlitis and perityphlitis during 89
Pregnancy, heart disease and.... 89
Pregnancy, Kiutsi-Malone urinary test for................... 185
Pregnancy, menstruation-resembling, during 393
Pregnancy, pyelitis of, with especial reference to its etiology 290
Pregnancy, Wassermann test in 393
Pregnant woman, energometric observations in the........... 88
Prostatitis and spermatocystitis, bacteriology of chronic....... 183
Prostatitis, metastatic 183
Proteins and their split products, ninhydrin color reactions of.. 276
Psychosis, menstruation and.... 86
Puerperal fever, the tubes in.... 394
Pulmonary affections and gout.. 78
Pulmonary edema, the prognosis of 120

PAGE
Purin metabolism, familial hydrops intermittens and..... 377
Pyelitis of pregnancy with especial reference to its etiology 290
Pyelocystitis in infancy, prognosis of 288
Pyelography, a source of danger in 183
Pyelography, observations on.... 182
Pyloric hypertrophy in newborn children 81
Pyonephrosis 392
Pyorrhea alveolaris, chronic bronchitis and asthma........ 169
Quinke's edema, pathogenesis of 159
Radiology of the digestive tract, some remarks on the underlying principles and the clinical value of 27
Reinfection in lues.............. 382
Relapsing fever, a new affection resembling 382
Renal activity during pregnancy, ·functional determination of... 89
Renal infections, non-tuberculous 392
Röntgenograms of the chest in tuberculosis, interpretation of. 279
Röntgen ray diagnosis of gas and gas infections 282
Röntgen ray examination in tuberculosis of infancy........ 74
Röntgen ray examinations in the diagnosis of cancer of the stomach, value of............ 283
Röntgen ray in lobar pneumonia in childhood 74
Röntgen ray localization of dysenteric ulceration 75
Röntgen rays in the diagnosis of gastric ulcer, duodenal ulcer and gastric cancer............ 316
Russo reaction 73
Salomon's albumin determination in the gastric contents........ 173
Scarlet fever, etiology of........ 161
Scarlet fever, tuberculin reaction in 383
Schick test 162, 163
Sclerema neonatorium 76
Secretion, gastric, examination of normal, by the fractional method 284
Secretions, myopathy and disorders of internal............. 278

PAGE
Secretion, pancreatic 82
Senile cases, diagnostic errors in 130
Serology and the diagnosis of syphilis 167
Sinus arrhythmia, clinical significance of 385
Sinus, injuries of superior longitudinal 84
Soldier's heart 170
Speculum-camera, a 289
Spermatocystitis, bacteriology of chronic prostatitis and........ 183
Spinal fluid syndromes of Nonne and Froin 287
Spirochetes, method of detecting 73
Spotted fever, Rocky Mountain.. 164
Sputum examinations, comparative, by means of the Ziehl-Neelson and the Kronberger methods 164
Stain, a new blood.............. 155
Sterility, fertility and........... 392
Stomach, acidity of the nursling's 80
Stomach, detection of pepsin and trypsin in the fasting......... 80
Stomach, disease of, Gluzinski's method in the diagnosis of.... 171
Stomach, mucormycosis of the.. 386
Stomach, occult hemorrhages in ulcer and carcinoma of the.... 173
Stomach of the nursling, acidity and pepsin digestion in the.... 186
Stomach, opaque meal shadows in diseases of the........... 80
Stomach, syphilis of the........ 174
Stomach, the significance of the tongue in the diagnosis of diseases of the.................. 23
Stomach, value of Röntgen ray examinations in the diagnosis of cancer of the.............. 283
Syphilis and aorta.............. 169
Syphilis and diabetes, latent..... 280
Syphilis, hereditary 168
Syphilis, hereditary, and spurs on os calcis 280
Syphilis, infectivity of cerebrospinal liquid in............... 181
Syphilis, luetin reaction in congenital 168
Syphilis of the stomach......... 174
Syphilis, serology and the diagnosis of 167
Syphilis with secondary involvement of heart and kidneys.... 168

PAGE
Syphilitic aortitis 170
Syphilitic nephritis, acute....... 169
Syphoning, the urine after gastric 87
Tachycardia, the mechanism of paroxysmal 79
Tension, hemoptysis due to high 79
Tetanus, locally limited......... 382
Throat, pneumococcal 281
Thymus disease in children..... 312
Thyroid disturbances, tonsilar endamebiasis and 73
Thyroid preparations, the effect of, on thyrodopaths.......... 376
Thyroid secretion, physiological activity of 375
Thyroid vessels, thyroid and circulatory changes following experimental ligation of the.... 158
Tongue, the significance of the, in the diagnosis of diseases of the stomach 23
Tonsils and chronic cervical adenitis 76
Tonsil as a portal of infection other than rheumatic, the..... 323
Tonsilar endamebiasis and thyroid disturbances 73
Tonsillitis, appendicitis as sequela of 83
Trypsin in the fasting stomach, detection of pepsin and....... 80
Tubes in puerperal fever, the... 394
Tuberculin reaction in scarlet fever 383
Tuberculosis, complement-fixation in 383
Tuberculosis, eye findings in pulmonary 166
Tuberculosis, genitourinary, diagnosis of 183
Tuberculosis, incipient apical pulmonary 165
Tuberculosis in infants, fever, an initial sign of................ 165
Tuberculosis, interpretation of Röntgenograms of the chest in 279
Tuberculosis in the male genital system, the mode of spreading of 184
Tuberculosis of breast.......... 280
Tuberculosis of infancy, Röntgen ray examination in............ 74
Tuberculosis of the cervical lymphatics 383

PAGE
Tuberculosis of the male genital system 87
Tuberculosis of the mammary gland 166
Tuberculosis, subfebrile temperature in the diagnosis of incipient pulmonary 382
Tuberculosis, the diazo- and urochromogen reactions as prognostic aids in pulmonary...... 279
Tumors, diagnostic elements of ponto-cerebellar 64
Typhlitis and perityphlitis during pregnancy, genuine 89
Typhoid carriers, search for..... 163
Typhoid fever, the Gruber-Widal reaction in persons vaccinated against 381
Typhus fever, diagnosis of...... 164
Typhus fever, investigations concerning 381
Ulceration, Röntgen-ray localization of dysenteric............ 75
Ulcer, duodenal, indications for operative interference in...... 91
Ulcers, gastric and duodenal.... 81
Urea excretion, rate of......... 154
Uremia 88
Urinary constituents in hepatic disease, excretion of certain.. 176
Urinary organs in infants, bacterial infections of the........ 186
Urine after gastric syphoning... 87
Urine, amylase in the blood serum and 276
Urine, detection of albumin in. 157
Urine, determination of nitrogen in 378
Urine in children, chronic retention of 181
Urine of normal children, ketones and Beta-oxybutyric acid in.. 154
Urticaria tuberosa of Willan... 286
Vaginitis in infants............ 395
Vision, disturbance of, from cerebral lesions 390
Vomiting of children, diarrhea and 285
Vulva, primary carcinoma of the 394
Weil's disease 381
Willan, urticaria tuberosa of.... 286
Wounds, bacteriology of septic.. 75
Wounds, gangrenous, bacteria of 75
Yellow fever, neutrophile leukocytes in malaria and.......... 74

INDEX OF AUTHORS

Page reference in bold-face type indicates a Special Article or General Retrospect; in ordinary type, an Original Abstract of an Article that has appeared in full elsewhere.

Abt, I. A., 385.
Addis, T., 154.
Ager, L. C., **205.**
Albu, A., 377.
Anderson, H. B., 83.
v. Angerer, K., 159.
Aravantinos, A., 376.
Asher, L., 375.
Ashner, P. W., 277.

Baetjer, F. H., 283.
Bandler, S. W., 293.
Barach, J. H., **218.**
Barton, E. A., 289.
Basch, S., **31.**
Bedell, A. J., 166.
Beer, E., 181.
Bernhard, A., 277.
Bigler, W., 88.
Bittorf, A., 375.
Blaivas, A. J., 378.
Boas, I., 173.
Bock, F., 83.
Bosch, S., 164.
Bridgman, E. W., 386.
Brünaauer, S. R., 162.
Bruns, 391.
Brunzel, H. F., 382.
Bucura, C., 185.
Bullock, W. E., 74.

Cabot, H., 392.
Callahan, F. F., 279.
Cameron, H. C., 278.
Campbell, W., 281.
Carlson, R. B., 84.
Carlson, H. E., 189.
Chambers, J. S., 276.
Churchman, J. W., 183.
Clarke, C., 163.
Climenko, H., 85.
Codd, J. H., 80.
Coerper, C., 157.
Cohn, J., 378.
Coles, H. C., 73.
Connell, F. G., 387.
Coolidge, L. H., 381.
Cornwal, E. E., **22, 233, 331.**
Corper, H. J., 279.
Corper, J., 382.
Courvoisier, H., 375.
Culver, H. B., 183.
Cutter, F. S., 185.

Damson, B. 163.
Dana, C. L., 180.
Danforth, W. C., 290.
Davis, G. G., 282.
Davis, J. E., 177.
Dean, H. R., 75.
Deaver, J. B., **91.**
Dennett, H. H., **143.**
Dorner, G., 80.
Dowd, C. M., 383.
Dreyer, G., 163.
Dugeon, L. S., 84.
Dunn, C. H., 74.
Durante, L., 280.

Einhorn, M., 82, 174.
Emerson, H. W., 276.
Emmert, J., 173.
Epstein, A. A., 277.
Epstein, J., **126.**
Epstein, S., **327.**
Eustis, A. E., 168.
Evans, F. A., 156.
Evans, J. S., 73.

Fahr, T., 182.
Falta, W., 292.
Fellner, O. O., 89.
Fielder, F. S., 282.
Findlay, L., 385.
Fleming, A., 75.
Floyd, C., 281.
Forbes, F. H., 393.
Fowler, R. S., **209, 214.**
Fraser, A., 287.
Frenkel-Tissot, H. C., 377.
Friedenwald, J., 283.
Friedman, G. A., **100.**

Frühwald, R., 181.
Fuchs, A., 389.
Furniss, H. D., 395.

Gaham, C., 81.
Gallant, A. E., **267**.
Gamble, M. F. H., 380.
Gardiner, H., 76.
Garrod, A. E., 292.
Gassot, 394.
Gatewood, 166.
Gerstmann, J., 377.
Geyelin, H. R., 158.
Gibson, A. G., 163.
Gibson, C. L., 172.
Gisel, A., 378.
Glasson, C. J., 75.
Goetsch, E., 159.
Goldberg, B., 391.
Goodhart, S. P., 85.
Gordon, A., **64**.
Gordon, M. B., 168.
Gradwohl, R. B. H., 378.
Griswold, D. M., 162.
Grossman, M., 165.
Grulee, C. G., 288.
Gruner, O. C., 156.

Hall, F. De H., 79.
Hall, J. N., 172.
Halliburton, W. D., 379.
Hammon, L., 169.
Hanes, F. M., 287.
Hart, T. S., **31**.
Hassman, D. M., 383.
Hays, H., **323**.
Hebold, O., 86.
Heidingsfeld, M. L., 167.
Heiman, H., 77, 85.
Heimann, 378.
Hertzler, A. E., 189.
Herz, M., 169.
Herzfeld, E. 176.
Herxheimer, G., 381.
Hess, A. F., 395.
Hess, R., 80.
Hicks, H. T., 393.
Hoffman, F. L., 188.
Holmes, G., 84, 390.
Hormstein, M., 90.
Horrax, G., 277, 278.
Howell, W. W., 77.
Howland, J., 155, 377.
Hügelmann, 83.
Hunt, J. R., 178.
Hutchinson, R., 285.
Hymanson, A., 72.

Jack, W. R., 384.
v. Jaksch, 178.
Jaschke, R. T., 89, 185.
Jastram, M., 376.
Jefferson, G., 386.
Jolly, P., 86.
Johnston, M. R., 154.
Jones, H. E., 72.

Kahn, M., 73.
Kaufmann-Wolf, M., 186.
Keller, H., 73.
Kerr, Le G., 168.
Kleeberger, K., 375.
Kocher, A., 376.
Kolmer, J. A., 162.
Konjetzny, G. E., 157.
Koplik, H., 281.
Kowalski, B., 382.
Kowitz, H. L., 186.
Krabbe, K., 390.
Kronberg, R., 186.

La Franca, S., 79.
Lanz, W., 376.
Larkin, J. H., 170.
Larson, W. P., 76.
Latch, E. A., 384.
Lee, R. I., 78.
Lehnhoff, H. J., **139**.
Lemann, I. I., 283.
Lemchen, B., 155.
Lenneberg, R., 383.
Leopold, J. S., 277.
Lerch, O., 1.
Lespinasse, V. D., 288, 289.
Levinson, A., 389.
Levy, I. H., **316**.
Levy, I. J., 170, **295**.
Lichtenstein, L., 382.
Leichtweiss, F., 164.
Lichtwitz, L., 83.
Lichty, J. A., **230**.
Liebers, 157.
Lister, W. T., 390.
Litzenberg, J. C., 290.
Logan, W. R., 388.
Lovett, R. W., 293.
Löwy, J., 389.
Ludlum, S. D. W., 278.

Macfie, J. W. S., 74.
Machwitz, H., 88.
Mackenzie, J., 170.
Mallory, F. B., 161.
Mapes, C. C., 175.
Marriott, W. McK., 155, 377.

Martin, A., 395.
Martin, A., 395.
Martin, W. B. M., 385.
Mason, H. H., 74.
Matko, J., 87.
Maybury, B. C., 84.
Mayer, A., 78.
McCarthy, W. C., 280.
McCouch, G. P., 278.
McIntosh, J., 74.
Medlar, E. M., 161.
Mellanby, E., 285.
Mendel, L. B., 277.
Meyers, M. K., 292.
Middleton, W. S., 73.
Moldowskaja, L., 388.
Moody, S. S., 162.
Moore, J. J., 393.
v. Moraczewsky, 176.
Moravek, A. J., 73.
Morris, R. T., 386.
Morrison, H., 377.
Morse, J. L., 166, 281, 383.
Morse, M., 185.
Moshage, E. L., 162.
Mott, F. W., 178.
Mouat, T. B., 75.

Nascher, I. L., **130.**
Neal, J. B., 160.
Nettle, A., **69.**
Neuda, P., 159.
Neuhof, H., 79, **98,** 158.
Nixon, J. A., 286.
Norbury, F. G., 282.

Ohly, 174.
Olmstead, M. P., 74.
Osborn, F. B., 277.

Paine, L., 164.
Paterson, W. S., 76.
Peabody, F. W., 155.
Perkins, A. E., 179.
Petersen, A., 89.
Plass, E. D., 291.
v. Podmaniczy, T., 170.
Pok, J., 393.
Pollitzer, H., 87.
Povitzky, O. R., 74.
Pulvermacher, 393.

Ramsay, J., 384.
Rehfuss, M. E., 284.
Reilly, T. F., **120.**
Resch, A., 157.
Reuben, M. S., 165.
Reynolds, E., 392.
Riedel, 387.
Riesman, D., 158.
Roberts, P. W., 179.
da Rocha-Lima, H., 381.
Rolleston, J. D., 280.
Rosenberg, M., 88, 154.
Rosenbloom, J., 386.
Rosenfeld, G., 376.
Rosin, H., 79.
Ryle, J. A., 381.

Saltykow, S., 79.
Sambergerm, F., 174.
Sargent, P., 84.
Schlaepfer, V., 382.
Schleicher, 177.
Schloss, O. M., 285.
Schütz, 174.
Scokalski, C., 381.
Secher, K., 162.
Sedgwick, J. P., 76.
Seidl, 171.
Shastid, T. H., 189.
Shattuck, G. C., 170.
Sheffield, H. B., **312.**
Shirdas, S. P., 169.
Silbergleit, H., 159.
Simmonds, M., 87, 159, 183, 184.
Singer, J. J., 279.
Smith, G. F. D., 292.
Smith, J. A., 73.
Snyder, J. R., 161.
Spencer, H. R., 289.
Stein, A., 379, 394.
Stein, H. E., **108, 116.**
Stern, Heinrich, 187.
Stochs, O., 276.
Stokes, A., 163.
Stokes, A. M., 381.
Stokes, J. H., 169.
Stoll, H. F., 77, 280.
Stone, W. S., 184.
Strasser, A., 164.
Strauss, A., 83.
Sturmdorf, A., **302.**
Swett, P. P., 280.
v. Szöllösy, L., 87.

Talbot, E. S., 284.
Teacher, J. H., 384.
Ten Broeck, C., 282.
Terhune, W. B., 390.
Teutschländer, O., 386.
Thorn, D. A., 389.
Thorn, L. T., 385.
Tilton, B. T., 175.

Torrey, J. C., 163.
Tugendreich, G., 395.

Underhill, F. P., 187.

Vander Hoof, D., **23**.
Veeders, B. S., 154.

Wachenheim, F. L., 81.
Wagner, A. W., 388.
Walker, E. W. A., 163.
Wallace, C., 286.
Wallis, G. F. C., 84.
Ward, H. B., 177.
Warthin, A. S., 280.
Watanabe, C. K., 154.
Webster, J. C., 290.
Wechsler, L., 73.
Weil, G., 375.
Weiland, W., 157.
Weinberg, 171.
Weinberger, W., **191**.
Weston, P. G., 180.
Wildbolz, 183.
Williams, B. G. R., **227**.
Williams, E. C., 72.
Wilson, U. F., 280.
Williamson, C. S., 379.
Windle, J. D., 171.
Witington, F. H., 163.
Worthen, T. W., 285.
Wulff, O., 173.

Zalo iecki, A., 181.
Zingher, A., 163.
Zoeppritz, H., 171, 173.
Zollenkopf, G., 382.
Zondek, 181.
Zondek, H., 391.

THE
ARCHIVES OF DIAGNOSIS
A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX JANUARY, 1916 No. 1

Special Articles

COMPARISON OF "DROP" PERCUSSION WITH THE METHODS NOW IN USE AND ORTHODIOGRAPHY

By OTTO LERCH
Professor of Diagnosis and Treatment, Tulane University of Louisiana,
Post-Graduate Department
New Orleans, La.

To determine form, position and size of the various organs of the body is a necessity in order to make a correct diagnosis. A change in size means in every instance structural disease; if too large it points to congestion, hypertrophy or dilatation, and if too small, insufficient development or shrinkage. A change in position means in most cases visceroptosis and functional disease, though in some instances, organic disease may displace the organs, besides altering their size and shape. Up to the time of the discovery of the Röntgen rays the physician had to depend solely on percussion and palpation to obtain these important data. Since then, X-rays are widely used for diagnostic purposes. They are not in reach of everybody on account of the apparatus needed and the expert work it takes to obtain satisfactory results; they cannot be used at the bedside and their employment is too expensive and requires too much time in general practice. Röntgen rays can never replace percussion and palpation for these reasons and the practitioner has to depend on them to make his diagnosis in most cases. Percussion is by far the more important of the two for this purpose and the literature on the subject is voluminous since Laennec introduced it.

Direct percussion, that is, the tapping of the body surface with finger or knuckel, has been almost universally replaced by intermediate percussion, and the methods in common use are the finger-finger percussion, the finger-pleximeter percussion and the hammer-pleximeter percussion. The principle does not differ, the sound produced by a stroke guides to outline an organ and to discover pathological conditions in chest and abdomen. The method has its drawbacks, as only a light and elastic stroke will give good results, and what is meant by that, depends on the judgment of the examiner. This differs and with it the sound produced. The interpretation of the sound also is individual and depends on training and acuteness of hearing. It is, therefore, very rare and an accident if two observers get the same results in a given case. Modifications have been suggested to make the method more accurate and uniform. I mention auscultatory percussion, medium percussion and Goldscheider and Ewald's threshold percussion. All depend on the sound produced by a stroke or tap, the latter more or less lightly made. The principle remains the same. The method is individual, active, a fine art, difficult to acquire. My method differs in principle. *I use a drop instead of a stroke.* Hammer and pleximeter are necessary to obtain good results. I use a hammer with a heavy steel head, a soft black rubber tip, light ebony handle and a thin ivory pleximeter.* A drop is passive and cannot be changed, and the sound produced is the same, no matter who allows the hammer to drop. The interpretation of the sound, more definite and delicate than the one produced by a stroke, is aided by palpation, also passive; the finest change of vibration produced by the falling hammer is at once felt by the hand that holds the pleximeter, as well as by the hand that holds the hammer, and the rebound changing with the changing vibrations is readily seen. *If not seen there is no drop, but stroke,* and the result likely not exact. Eye, ear and sense of touch unite to give the result which in every instance is correct. *The method is one of precision.* The height from which the hammer drops is immaterial as one sense compensates the other and the result remains the same. The hammer lightly held is tossed up with a light wrist movement and allowed to drop from 2 to 5 c.m. upon the pleximeter. Either height may be used, or any other, one controls the other; vibrations pass through the whole chest, no matter how light the hammer drops. The method is *mechanical, easy to learn, and gives uniform results in everybody's hands.*

---

*Albert Schroeder and I. L. Lyons, of New Orleans, sell these hammers.

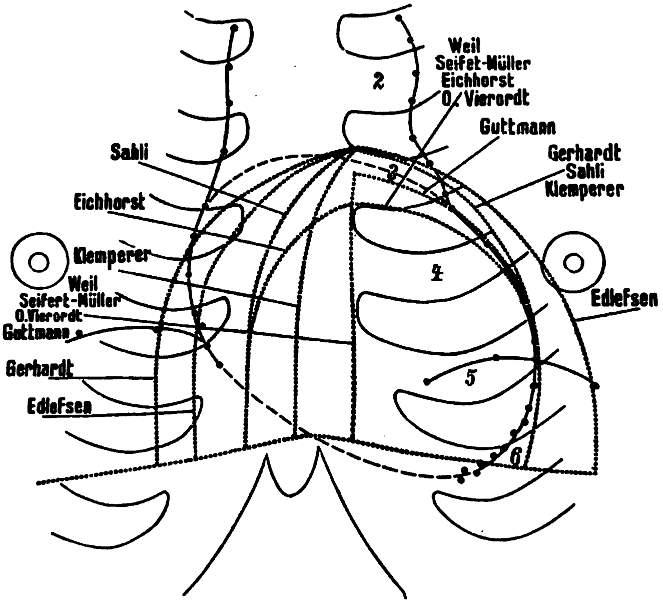


FIG. I

Orthodiagram of the normal heart in normal position of a man 24 years of age. The borders of the relative heart dulness are drawn in the skeleton of the thorax as they are given by a number of text-books in use. (After Moritz, Deutsches Archiv für Klinische Medizin, Oct. 24, 1906.)

Figure I shows the results of percussion obtained by various observers with the methods in use, compared with the orthodiagram. The great difference of opinion of the various observers as to the size of the heart is marked. The position of the domes of the diaphragm is incorrect; the heart rests between them and upon the fixed diaphragm. Notwithstanding, a diagnosis can be made if the examiner masters the method. The error always the same, is relative only.

Percussion has stood the test of time. Every physician in active practice uses it daily. Only in recent years, however, more delicate results have been obtained with it. Ebstein and Reiss claimed to be the only clinicians that up to 1906 mapped out the whole anterior surface of the heart. Since then, many others have succeeded, and Ewald and Goldscheider were able to differentiate between liver dulness and heart dulness with their method. Yet the great difference in results obtained by teachers is confusing and discouraging to the student, who is apt to place but little faith in a method which takes so much time and patience to master, and with all, is not accurate and delicate.

No improvement has been made since Goldscheider and Ewald introduced their method of threshold percussion, and notwithstanding better results are obtained in recent years than formerly, they are probably due to the fact that since the discovery of the Röntgen rays, radiography is used in every clinic to control the results of percussion.

Wood makes this statement:*

"When studying physical diagnosis as a student, one of the things difficult for me to understand was percussion. As I talk to students in the medical schools now, I am told by them that percussion is one of the hard things for them to learn, and that it means the least to them of the physical signs."

Wood's experience is that of most clinical teachers. The difference in results obtained by various observers is due to different conditions under which they percuss (character of stroke and pressure of the pleximeter) and to a certain difference of the note which they have accepted for their determination.

Fig. II. Hirschfelder thinks that in persons with round, narrow chests, or in persons with large hearts that almost fill the left half of the thorax, it is impossible to determine the true border. The curve of the ribs follows the wall of the left ventricle, and the latter may remain near the chest wall throughout the axilla. The outer border of dulness may be obtained not over the apex, but over the posterior wall of the left ventricle. If my method is used, this is not so. The thickness of the heart, as well as its anterior surface, can be accurately made out in every case, no matter what size and

form of chest and heart. That is O—O and p'—p' can be made out correctly.

---

*"Percussion of the Lungs," Jour. A. M. A., Oct. 17, 1914.

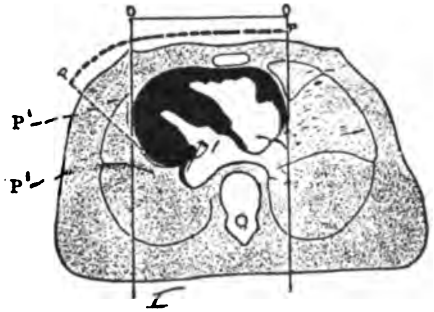


FIG. II

Diagram to show the cause of unavoidable error in percussion of the cardiac outlines. P - - - P, outline in percussion; O - - - O, orthodiagraph outline. (After Hirschfelder, Diseases of the Heart and Aorta, p. 147.) "P'P' thickness of the heart can be determined by drop method."

If the posterior wall of the heart comes close to the chest wall, the heart beat may be conveyed and taken for the apex beat (diffuse apex beat and double apex beat). The dulness of the anterior surface of the heart can be differentiated from the dulness of the posterior surface by a zone of resonance that separates the two, the thickness of the chest wall and space between it and the anterior surface of the heart P' O.

It is important to know the thickness of the heart, hypertrophy may progress along the sternodorsal diameter and leave the examiner under the impression that there is nothing abnormal. Oestreich calls these hearts kidney hearts.

Figs. III and IV. Simon tested Goldscheider and Ewald's method on the cadaver and found it correct. This method can only be used in absolutely quiet rooms.

Fig. V. Dietlen, a pupil of Moritz, examined the heart of 231 patients with finger-finger percussion, medium stroke and firm pressure of the pleximeter. He comes to the conclusion that in from two-thirds to three-fourths of all cases the heart can be mapped out correctly, that is percussion corresponds to the orthodiagram allowing 0.5 c.m. difference.

De la Camp finds 85 per cent. correct, allowing a margin of 0.75 c.m., and Moritz finds 86 per cent. of the right border and 70 per

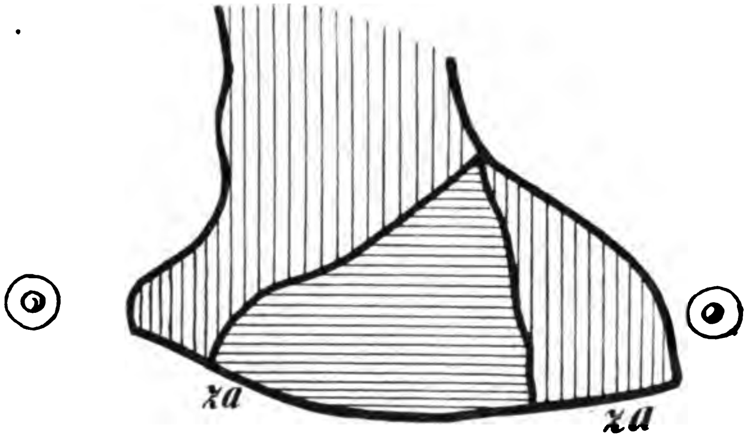


FIG. III

Man 57 years old. Dilatatio et Hypertrophia cordis, Hyperemia and edema pulmonum. (After Simon.)

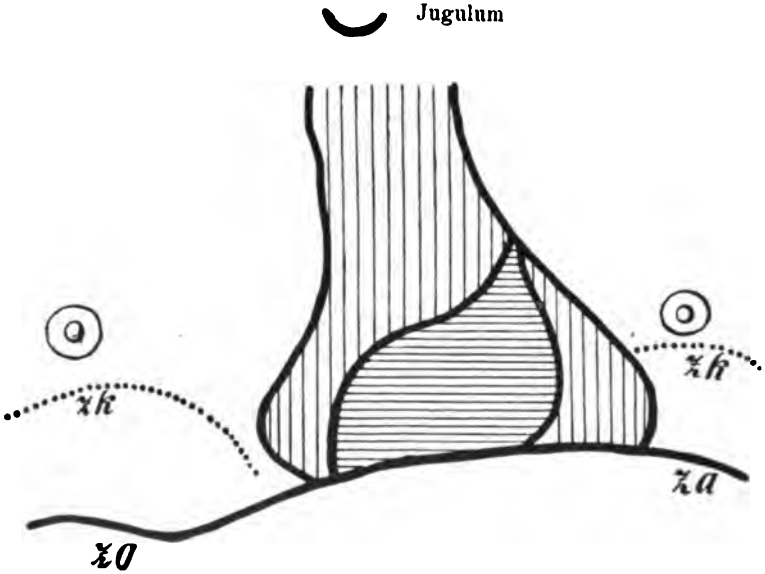


FIG. IV

Man 28 years old. Myocarditis parenchymatosa; Pleuropneumonia fibrinosa duplex; Pleuritic exudation, left more than right. (After A. Simons.) (Threshold percussion method of Ewald and Goldscheider, Deutsches Archiv, Klinische Medizin, 1906.)

cent. of the left border correct, allowing 0.5 c.m. difference between his percussion and the orthodiagram.

This shows that the experts map out the heart from two-thirds to three-fourths of all cases correctly, allowing an error from 0.5 c.m. to 0.75 c.m. The general practitioner falls far short of these results.

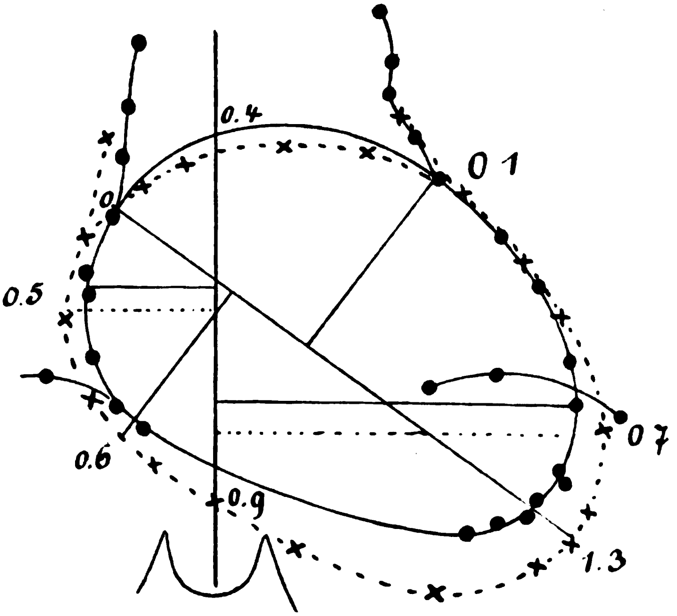


FIG. V

The numbers give the different measures in C. M. in eight places between the orthodiagrams .-.-. and the border of the heart xiii xiii x mapped out by percussion. Medium percussion, method of Moritz. (Dietlen, Op. cit., p. 237.)

The same is true for the liver and lungs. Few make out the whole liver and it is exceedingly difficult for most to outline the lower border and gallbladder, especially when the abdomen is distended. The same difficulty is met with when determining the borders of the lungs to discover pathological conditions in them. When physicians meet in consultation, they usually differ, and the X-rays have to be resorted to in cases where this is possible. The spleen can be mapped out, but by few, unless so much enlarged that it can be felt. Many claim that percussion is useless to determine the border of the spleen, and all agree that size and form of the kidneys cannot be determined by percussion.

To prove that my method gives accurate results, I have used it on the cadaver jointly with Professor Oestereich, of Berlin, and found it in every instance correct. More than this, it shows anatomical conditions, which previously have not been made out,—the large vessels, the free portion of the pericardium, the thymus from infancy to old age, the right and left auricle, the whole anterior surface of the heart and its thickness, the domes of the diaphragm and its fixture, the borders of the lungs and slight pathological changes in them, the spleen, and the size and form of the kidneys. Kidneys are found movable far more frequent than is usually supposed, and in such cases the projection upon the surface of the body shows a small kidney or an oval according to the angle under which the kidneys rest to the surface. If the patient is directed to turn himeslf around when in the recumbent posture, the kidneys change position and with it the dulness. To obtain their correct size in these cases, repeated examinations have to be made, and especially after the patient has been in bed for a few days. *In the recumbent posture, movable organs return to their normal position and this is the reason why the pathologist so frequently disagrees with the clinical findings.*

The following charts and histories show the results obtained with my method on the cadaver and on the living. In former publications on this subject, I have proposed the name of "Drop Percussion," being well aware of the fact that this is a misnomer and pointing out that the "Drop" is the essential feature of the method and that it has nothing in common with the methods of "Percussion" (stroke). Drop-stroke is an impossible combination. In fact the method differs more from percussion, than does palpation. It is more correctly called "Drop Method." Still, few practitioners think of this and have only in mind that percussion is a method to determine form, size and position of organs. I, therefore, thought it best to retain the term to aid the introduction of the method.

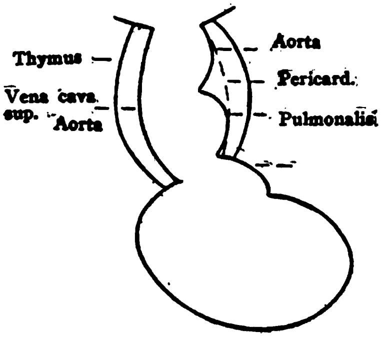


FIG. VI
Male child. Died of severe bronchitis and intestinal catarrh.

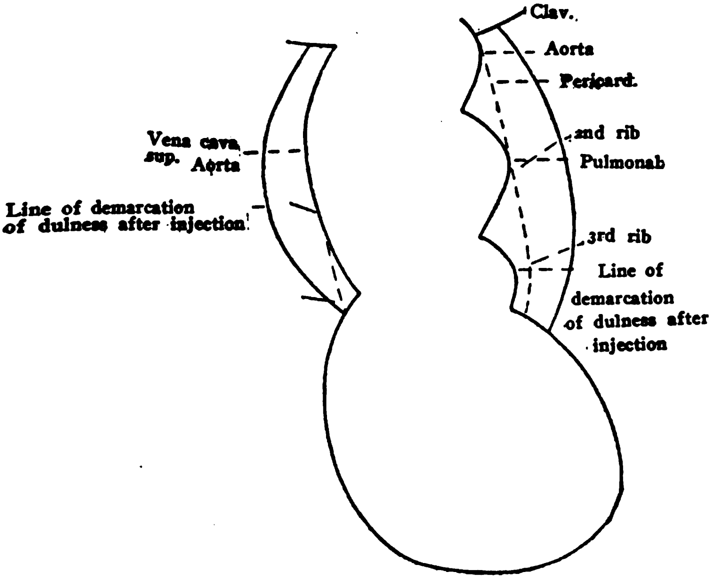

Clav.
Aorta
Pericard.
2nd rib
Pulmonalis
3rd rib
Line of demarcation of dulness after injection
Vena cava sup.
Aorta
Line of demarcation of dulness after injection

FIG. VII
Man 48 years of age. Died of general tuberculosis. Thymus after injection.

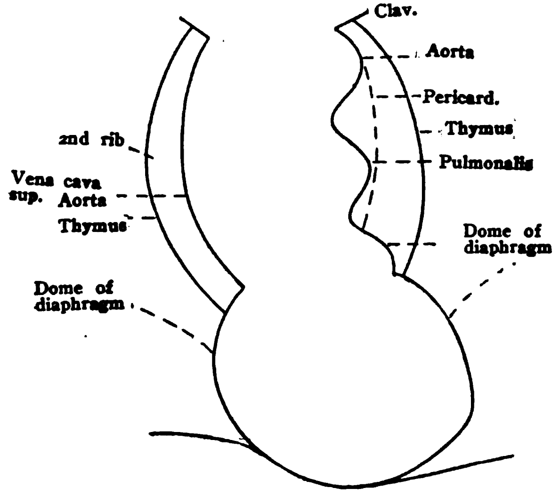


FIG. VIII

Woman 26 years of age. General Tuberculosis. Percussion after injection of 2 ltrs. of tormalin solution in the femoral artery. Percussion of the thymus on the cadaver is only possible after such an injection. A few hours after death the gland is free of blood and cannot be percussed.

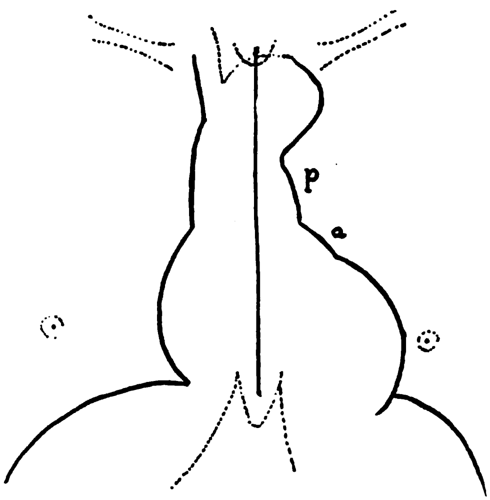


FIG. IX

Man 45 years of age. With thickened arteries and marked arterial hypertension. (Orthodiagram after H. Wessler.)

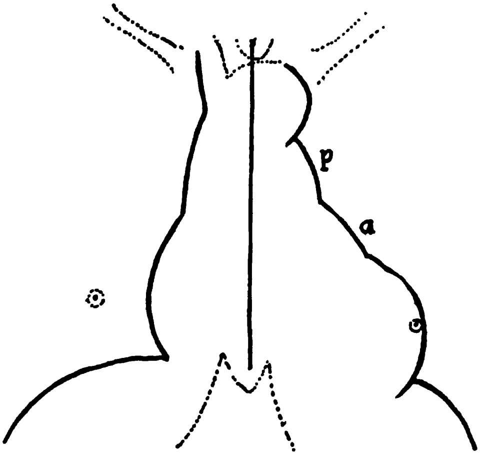


FIG. X

Same patient one year later with symptoms of heart failure. (Orthodiagram after H. Wessler. Am. Med., Oct., 1913.)

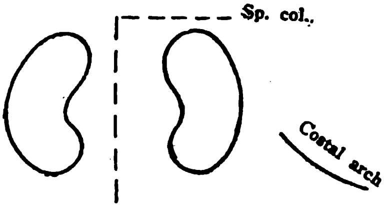


FIG. XI

Kidney dulness in a boy 8 months old who died of severe bronchitis and intestinal catarrh.

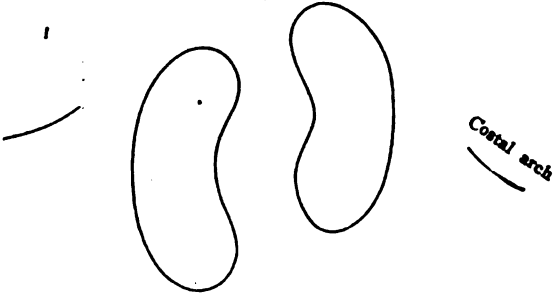


FIG. XII

Woman 60 years old. Movable kidney. Kidneys small, contracted. Autopsy: advanced arteriosclerosis and hemorrhages into the left cerebral hemisphere. Heart, left ventricle hypertrophied, and cirrhotic kidneys. "Percussion" correct.

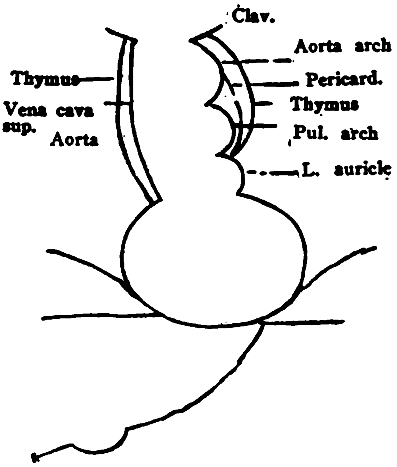


FIG. XIII

Miss S. Mitral insufficiency, enlargement of r. heart, vessels large, March 4, 1911. Patient 5 feet 4 inches. 120 pounds.

MITRAL INSUFFICIENCY

Miss S. Fig. XIII. 28 years of age, had in April, 1907, acute articular rheumatism, during which she contracted an endocarditis, leaving her with an insufficiency of the mitral valve.

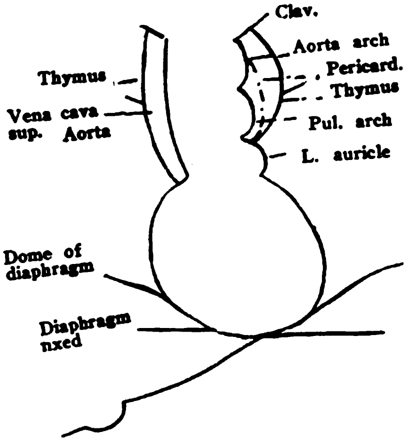


FIG. XIV a

Mrs. V., 45 years of age. 5 feet 6 inches. 145 pounds. Dec. 28, 1912.

She was examined Jan. 28, 1914, complaining of shortness of breath after exertion and of getting tired.

Heart, loud systolic blow, accentuation of second pulmonary sound and dulness of right heart enlarged as shown in the chart. Kidneys normal in size, urine nothing abnormal, all other organs normal, no edema.

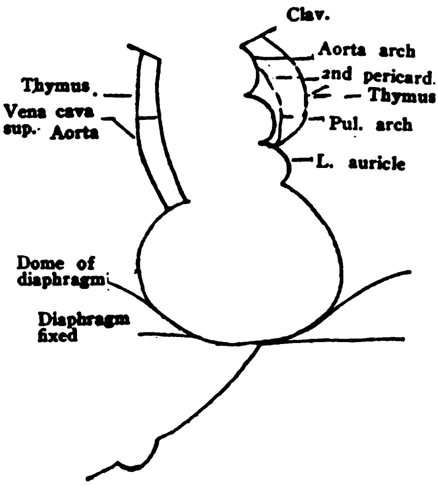


FIG. XIV b
Same patient November 3, 1914. Relative insufficiency of mitral valve.

DILATATION OF HEART WITH RELATIVE INSUFFICIENCY OF MITRAL VALVE

Mrs. V. Figs. XIV, a and b. Examined June 31, 1912. 45 years of age. Born in New Orleans, 5 feet, 6 inches, weighs 145 pounds.

Patient complains of fullness after eating and sour eructations, headache, backache, nervousness and chronic constipation.

Pulse 76, arteries palpable. Blood-pressure 138.

Heart sounds pure, lungs free, liver border hard and sharp.

Urine free of albumin and sugar. Sp. G. 1020. React. acid.

Kidneys both movable, normal in size; dulness 10 by 4 c.m.

Patient was re-examined, Nov. 3, 1914. She complains of pain in the back and joints, indigestion and disturbance of vision, describes it as if she had a fine film over both eyes, like cobwebs.

Pulse 80, blood-pressure 155.

Heart soft systol. blow at apex, right heart enlarged.

Liver border can be felt full and hard.

Eyes normal. Chart shows XIV, heart normal, XV b. 2 years later heart dilated, especially the right ventricle returned to the normal under treatment.

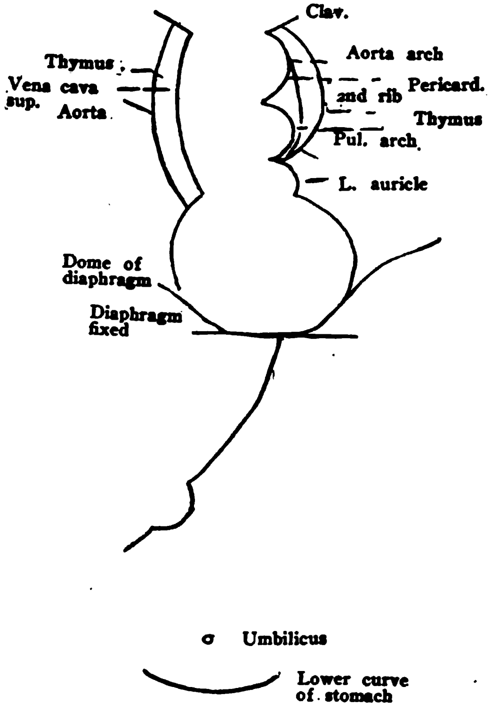


FIG. XV a

Mr. R. A. F. 5 feet 10 inches, 260 pounds. Dilatation of the heart; small thymus, as usual in the above. Oct. 18, 1914.

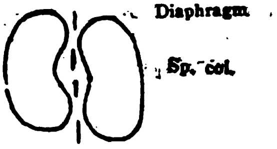


FIG. XV b

Mr. R. A. F. Oct. 18, 1914. Kidney dulness large. Returned to normal size after treatment 10 cm. by 4 cm.

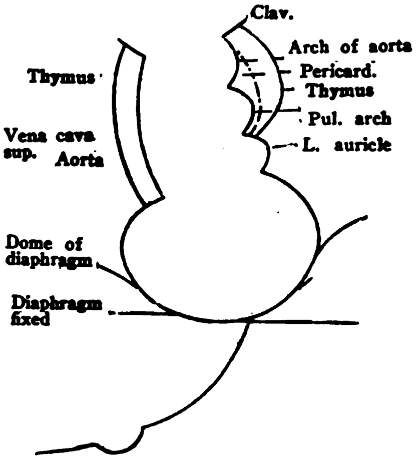


FIG. XV c

Mr. R. A. F. Same patient. Oct. 23, 1914. Heart reduced in size. Vessels less bulging.

OBESITY AND DILATATION OF HEART

R. A. F. Figs. XV a, b, c. 56 years of age, lumberman, does not take alcohol; is a moderate smoker. Has had yellow fever and malaria, no venereal diseases.

Complains of dizziness and shortness of breath.

The color of his face is slightly cyanosed. He is 5 feet 10 inches high and weighs 251 pounds. Blood pressure 135, pulse 96 regular, arteries compressible.

Lungs, fine, moist râles over base.

Heart sounds muffled.

Liver border full and smooth, not hard.

Heart dilated and kidneys large, due to congestion.

Urine changes in quantity and color. Is free of albumin, sugar and casts.

First examined Oct. 13, 1914.

Re-examined Oct. 23, 1914.

The chart shows the improvement.

Patient died December, 1915, acute dilatation of the heart.

The patient had stopped all treatment after first improvement, October, 1914, and died two days after he returned to New Orleans for business reasons in December, 1915. He had attended the theater the evening before he died.

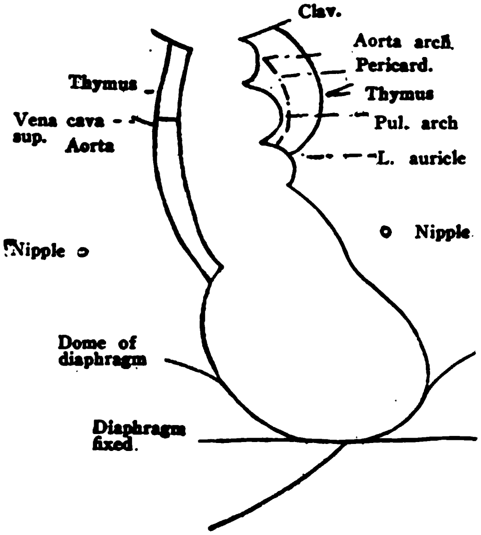


FIG. XVI a

Dr. P. examined, Oct. 24, 1914. Dilatation and stretching of a movable heart. Patient 5 feet 4 inches, 124 pounds. The dent in the curve of the left ventricle is due to sclerosis.

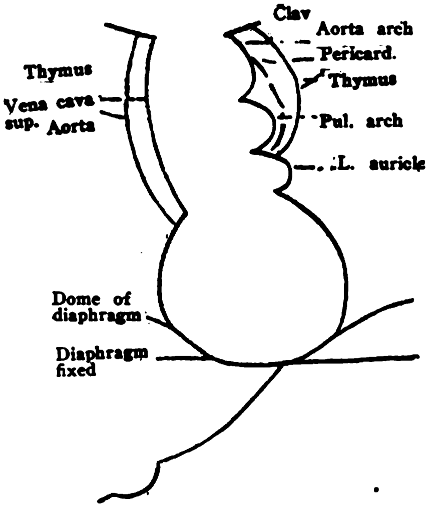


FIG. XVI b

Same patient re-examined. Nov. 10, 1914. Improvement marked.

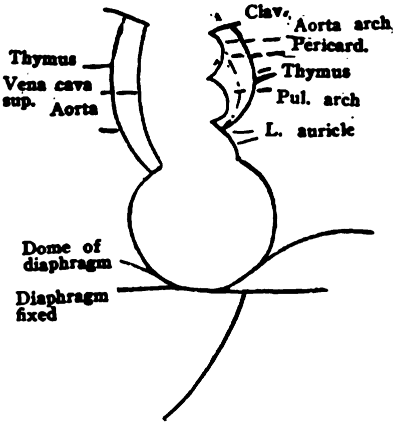


FIG. XVI c

Same patient, Nov. 21, 1914. Entire change of position, form and size of heart and vessels.

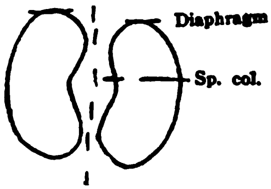


FIG. XVI d

Same patient. Kidney dulness small. 8 cm. by 3 cm.

DILATATION OF MOVABLE HEART AND HEART BLOCK GENERAL SCLEROSIS

Dr. A. P. Figs. XVI, a, b, c, d. Examined Oct. 24, 1914. 49 years of age, born in Cuba, physician. His mother died at 60 of arterio-sclerosis, two brothers died of heart disease at middle age.

Patient does not use alcohol in any form and has given up smoking for several years. He was formerly a moderate smoker. No syphilis. One year ago he had an attack of grippe; one month ago, due to an error in diet he believes, commenced to suffer with pain in stomach and bowels, sour eructations, palpitation, shortness of breath and cough, expectoration tinged with blood. He was treated by a stomach specialist in Cuba.

He is 5 feet 7 inches in height and weighs 127 pounds. Eyes prominent, arteries, tortion and hard, pulse small, 40 at the wrist; intermittent; blood-pressure 220. Heart, loud rasping systolic blow. A number of beats not conveyed to wrist. He says that previous to this attack his pulse was 65 and regular.

Right kidney palpable. Urine contains 4 per cent. of albumin and a few hyaline casts.

Re-examination Nov. 2, 1914.

Pulse 80 at wrist, intermittent, every beat conveyed. Murmur less rasping, blood-pressure 180. Entire change of position of form and size of heart and vessels. Urine free of albumin and casts. The chart shows the progress of improvement corresponding to his general health.

Re-examination Nov. 26th shows further improvement.

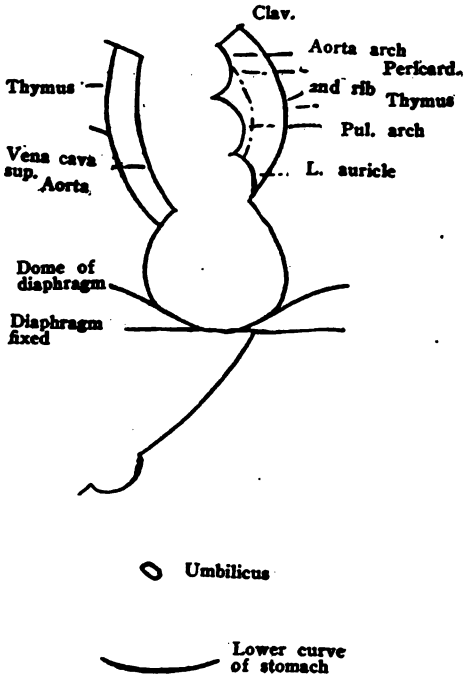


FIG. XVII a

Mrs. R. Cl. examined Nov. 15, 1914. Severe bronchial asthma enteroptosis. Large thymus always found in such cases. Thymus is distended with blood.

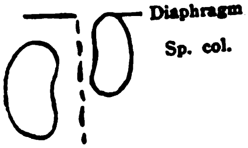


FIG. XVII b

Mr. R. Cl. Movable kidney. The kidneys stand under an angle to the surface. This and similar forms of kidney dulness are frequent in enteroptosis.

SEVERE BRONCHIAL ASTHMA, ENTEROPTOSIS AND LARGE THYMUS AS USUAL IN SUCH CASES

R. Cl. Figs. XVII, a and b. Examined Nov. 15, 1914. Electrician, 32 years of age. Does not use alcohol or tobacco in any form.

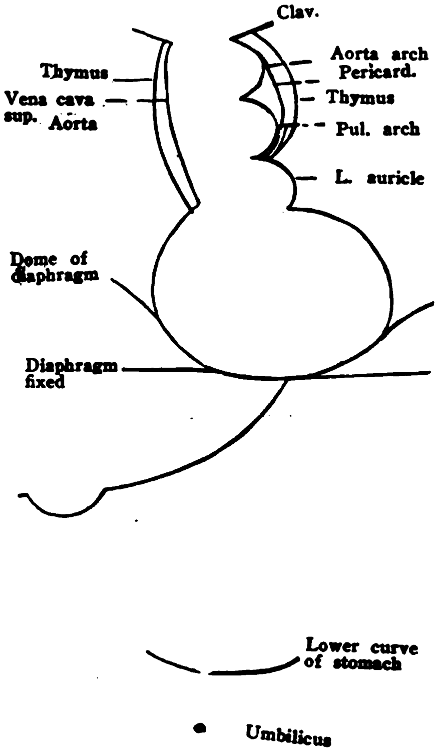


FIG. XVIII a

Mr. L. C. B. Oct. 12, 1914. Albuminuria and enteroptosis.

One brother died of tuberculosis. Patient has had a light attack of grippe. No infectious diseases. Has suffered from hay fever and asthma for seven years. For the last four months had to quit work on account of the severity of the asthmatic attacks. Patient is an enteroptotic, both kidneys are movable and lower curvature of stomach is handwide below navel.

Heart is small, soft systolic blow at apex, especially marked in recumbent posture.

Urine shows nothing abnormal.

Chart shows the form of displaced kidneys as frequently found in enteroptosis.

Thymus large—always in asthmatics, that is, in the many cases I have examined.

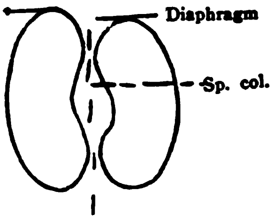


FIG. XVIII b

G. Le B. Nov. 22, 1914. Kidney dulness normal. 10 cm. by 4 cm. Only obtained after repeated examinations. The examination shows usually the dulness of displaced kidneys.

ALBUMINURIA AND ENTEROPTOSIS

G. Le B. Figs. XVIII a, b. 20 years of age, bank clerk, does not use alcohol or tobacco. Father died at 56 of Brights' disease, his mother is living and healthy. He had typhoid fever as a child.

Complains of pain in the back and of getting tired after standing up. Was examined for life insurance six months ago and was refused on account of albumin found in his urine.

He is 5 feet 8 inches high and weighs 144 pounds.

Patient is very nervous.

Pulse 90, regular, arteries contracted.

Right kidney palpable, both movable. Urine contains albumin only in the afternoon. No casts or cells. Patient is standing at his desk all day. First examined Oct. 12, 1914.

Kidney dulness, normal in size.

Albumin probably due to congestion.

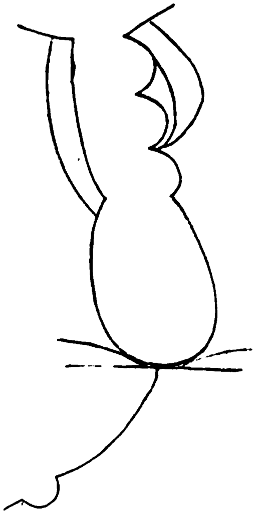


FIG. XIX

Mrs. H. examined March 21, 1915. The movable heart is frequently found round and small. Diaphragm depressed.

VISCEROPTOSIS AND DROP. HEART

Mrs. H., examined Nov. 21, 1915. Housekeeper, 45 years of age, mother of 4 children. She complains of severe digestive disturbances, of frontal headache and pain end of spine (pseudo coxalgia). Patient is 5 feet 8 inches high and weighs 117 pounds.

Pulse regular 90, arteries contracted, not palpable after pressure.

Blood-pressure 105.

Lungs free.

Heart soft systolic blow at apex.

Splashing over stomach 4 hours after light breakfast (coffee and toast).

Liver border palpable, hard and sharp.

Kidneys movable.

Urine nothing abnormal.

Stomach contents after test breakfast general acidity, with particles of toast and some mucus colorless liquid.

TWO VARIETIES OF PALPATORY PERCUSSION

By EDWARD E. CORNWALL
Brooklyn–New York

The two varieties of palpatory percussion which are here described are called by the writer "Feel" percussion and "Punch" percussion, respectively.

Feel percussion is performed as follows: With the tip of the finger of the right hand, the finger being bent as if for playing the piano and held loosely at the metacarpo-phalangeal articulation, and with motion mostly at the wrist, a series of very light touches or taps are made rapidly along a line on the surface of the body. Between these touches or taps the finger is raised very slightly from the skin, just enough to separate the touches or taps from each other; and no sound is made by the impact of the finger tip on the skin. The operation looks like feeling for something; and that is what it is. What is felt for is a sensation of difference in density, which informs the examiner of the presence of solider masses. This feel percussion is of particular value in mapping out the cardiac area, the hepatic area, the lower border of the stomach (where the wall of the viscus dipping in presents a narrow area of increased density to the examining finger which is "feel-percussing" over the surface of the abdomen), the inner border of the cecum and ascending colon, the lower border of the transverse colon, and the splenic area.

In *Punch percussion* advantage is taken of the fact that the pressure sense responds to a sudden impact more readily than to a slowly applied pressure of the same degree of tension (as in ballottement). It is performed by the middle finger of the right hand partly flexed at the metacarpo-phalangeal articulation and held rigidly straight, which is thrust vertically and with considerable force into the region to be examined. This method of palpatory percussion is particularly useful in examination of the abdomen for deep seated masses. The suddenly punched in stiff finger can sometimes appreciate the presence of a deep seated neoplasm or a displaced kidney which might escape being sensed by the slower pressure of ordinary palpation.

THE SIGNIFICANCE OF THE TONGUE IN THE DIAGNOSIS OF DISEASES OF THE STOMACH

BY DOUGLAS VANDER HOOF
Professor of Medicine in the Medical College of Virginia
Richmond, Va.

Every examination of the digestive organs may well begin with an inspection of the buccal cavity. The appearance of the tongue, however, gives very little evidence of real value, and is certainly subordinate in importance to the condition of the teeth, the tonsils and the pharyngeal wall. The tongue is in no sense the mirror of the stomach, and there is no direct relation between the coating of the tongue and affections of the stomach. The writer knows of only one condition of the tongue from which any forecast may be made of the gastric secretions, and that is the bald, red and often glazed tongue seen in many cases of gastric anacidity, often erroneously attributed to conditions of the stomach characterized by an excessive secretion of hydrochloric acid.

THE CAUSES OF COATED TONGUE

One very common cause of coated tongue is nasal obstruction. This occurs in an aggravated form in the individual who sleeps with his mouth open and awakens in the morning with a heavily coated tongue. The mouth is not intended as a respiratory passage, and any interference with the normal manner of breathing brings about an artificial condition of which one symptom is the furred, or coated, tongue. It is just in this way, moreover, that we can account for the coated tongue seen in almost every individual who smokes regularly.

In fevers from any cause there is more or less congestion of the mucous membranes of the nasal passages and the furred tongue is in part an expression of this obstruction to the normal breathing. In recent years the routine application of the "mouth toilet," by which the mouth and tongue are cleaned with a suitable mouth wash after every feeding, has done away with the coated appearance of the tongue, described by older medical writers as characteristic of one fever or another. One "variety" of coated tongue arises in cases associated with an accompanying superficial glossitis —the so-called "strawberry tongue"—seen most frequently in the early stage of scarlet fever, but not pathognomonic of this disease. This appearance of the tongue is due to the presence of a whitish fur through which project the greatly swollen and bright-red fungiform papillae. The same condition of the tongue, minus the fur, gives rise to an appearance called the "raspberry tongue."

Another frequent cause of coated tongue is absence of friction. Ill patients whose diet is confined to liquids generally have a coated tongue, which often disappears when solid food is begun. Insufficient mastication also plays an important rôle in causing the furred tongue of persons who eat too hurriedly or bolt their food. The posterior third of the tongue is constantly found coated in many normal individuals, and this is attributed by MacKenzie to the fact that in these cases the palate is high and arched so that the tongue does not come into contact with the roof of the mouth.

A third, and perhaps most common, cause of coated tongue is to be found in a perversion of the salivary secretion. The saliva is furnished by three sets of glands—the parotid, the submaxillary and the sublingual—to which is added the secretion from a number of small glands, belonging both to the serous and mucous types, which are imbedded in the mucous membrane of the mouth and tongue. The secretion from the submaxillary and sublingual glands is thick and mucilaginous, while that from the parotid is thin and serous. Furthermore, the salivary glands have a double nerve supply, cerebral and sympathetic, and the chemical composition of the secretion varies, not only upon alternate stimulation of these two different sets of nerve fibers, but also with the strength of the stimulation employed.

It has been shown that the flow of saliva is normally a definite reflex with a secretion center in the medulla oblongata, and as such this center may be affected by stimuli from various sources. Physiologists have demonstrated that this secretion center, and through it the salivary glands, may be called into activity by stimulation of the sensory fibers of the sciatic, splanchnic, and particularly the vagus nerves. So, too, various psychical acts, such as the thought of savory food and the feeling of nausea preceding vomiting, may be accompanied by a flow of saliva. Finally the medullary center may be inhibited as well as stimulated. The well known effect of fear, embarrassment, or anxiety, in producing a parched throat may be supposed to arise in this way by the inhibitory action of nerve impulses arising in the cerebral centers.

Thus it happens that most sick individuals have a coated tongue. This symptom appearing in a person who has previously possessed a clean tongue, merely indicates that there is something wrong in the economy. A careful examination may reveal the cause, and the appearance of the tongue may prove a guide as to the progress or retrogression of the ailment. As it often happens, however, many individuals have a coated tongue but are not aware of the fact until, feeling indisposed, they examine it in a mirror. Carrying this idea a step further, these same individuals after their recovery, whether it be due to a purgative or any other form of treatment, fail to examine themselves and so determine the continued presence of the coated tongue.

In the diagnosis of diseases of the stomach, practically no significance can be attached to the appearance of the tongue. Many years ago Boas emphasized the fact that a coated tongue may be seen in patients who enjoy a splendid appetite and an excellent digestion, and that, on the other hand, patients are found who suffer for weeks or months from anorexia and digestive disturbances, but whose tongues are perfectly clean. In spite of the increasing appreciation of the very subordinate position of the tongue in diagnonosis of gastrointestinal disturbances, most writers continue to state that the tongue is apt to be clean and red in gastric hyperacidity, and coated or foul in subacidity cases.

As a matter of interest the writer has reviewed the histories of 1500 consecutive patients on whom gastric analyses have been made, and has noted the accompanying description of the tongue. The results are shown in the following table:

Condition	Anacidity		Subacidity		Normacidity		Hyperacidity	
of the	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
Tongue	186	12.4	317	21.1	650	43.3	347	23.1
Not mentioned	49	....	55	....	102	....	50	....
Mentioned	137	....	262	....	548	....	297	....
Clean	48	35.0	94	35.9	192	35.0	81	27.3
Coated	89	65.0	168	64.1	356	65.0	216	72.7

The interesting fact is thus made clear that the same proportion of coated tongues, approximately 65 per cent., was found in patients having either normal gastric acidity, subacidity or anacidity, while cases of gastric hyperacidity showed a slightly higher relative number of coated tongues, namely 72 per cent. Investigations by Mueller and Fuchs (1898 and 1900) showed that 62 per cent. of apparently healthy people had coated tongues. In the writer's series the slightly higher percentage of coated tongues in hyperacidity cases probably has no significance. It is believed that a larger series of cases will show about the same proportion of coated tongues in both hypoacidity and hyperacidity, demonstrating again that the tongue is in no sense the mirror of the stomach.

THE TREATMENT OF COATED TONGUE

As suggested by the preceding remarks, treatment is either local or constitutional, or both. The essential thing is the attempt to remove the cause if this can be done. The futility of treatment directed toward real or supposed conditions of the stomach (or liver and bowels) is apparent, and has been for several generations past. As long as fifty-five years ago Oliver Wendell Holmes emphasized this fact and advised a unique, but practical method of directly cleaning the tongue. His remarks on this subject may well be repeated:

"One practical hint may not be out of place here. It seems to be sometimes forgotten, by those who must know the fact, that the tongue is very different, anatomically and physiologically, from the stomach. Its condition does not in the least imply a similar one of the stomach, which is a very different structure, covered with a different kind of epithelium, and furnished with entirely different secretions. A silversmith will, for a dollar, make a small hoe, of solid silver, which will last for centuries, and will give a patient more comfort, used for the removal of the accumulated epithelium and fungus growths which constitute the 'fur,' than many a prescription with a splitfooted ℞ before it, addressed to the parts out of reach."

REFERENCES

J. MacKenzie—Symptoms and Their Interpretation. 2d Ed. Paul B. Hoeber, 1913.

W. H. Howell—American Text-Book of Physiology. 2d Ed. Vol. I., W. B. Saunders & Co., 1901.
Mueller, and Fuchs—Quoted by P. Cohnheim—Diseases of the Digestive Canal. English Translation. J. B. Lippincott Co., 1909.
O. W. Holmes—Currents and Counter-currents in Medical Science—Medical Essays. Houghton, Mifflin & Co., 1899.

SOME REMARKS ON THE UNDERLYING PRINCIPLES AND THE CLINICAL VALUE OF RADIOLOGY OF THE DIGESTIVE TRACT

By SEYMOUR BASCH
Clinical Professor of Medicine, Fordham University; Attending Physician, Lebanon Hospital
New York

In recent years, more especially within the last two or three, rapid advances have been made both in the technic and in the interpretation of radiology of the digestive tract. This progress is largely due to the intelligent coöperation of the manufacturer, the radiographer and the clinician. The modern transformer, the intensifying screen, improved tubes and very rapid photographic plates have made possible far better radiographs today in a fraction of a second than were produced formerly in five or ten seconds. Röntgenology, previously left to the electrician, has become a well-established medical specialty through the entrance into the field of physicians with good technical and clinical training, who have not failed to take advantage of the better facilities offered them. This has led to the development in this country of the "positive or direct" method of demonstrating anatomical lesions on numerous serial plates, generally regarded here as a material advance over the "indirect or inferential" method, still largely employed in Europe, in which main reliance is placed on ocular or fluoroscopic examinations, with only an occasional plate.

As one of the clinicians, who have learned to recognize fully the value of radiography in this branch of medicine, I have found that both methods have their special advantages and that they should be combined in the study of each case. However, before radiography is resorted to at all, an attempt should be made to form a diagnosis through the approved clinical and laboratory methods. When a diagnosis is impossible or uncertain by these means alone, or where the choice between medical and surgical treatment remains in doubt, röntgenological studies should be undertaken. In every instance fluoroscopic examination should be combined with the radiographic.

The principles underlying röntgenography are identical for all portions of the digestive tract. The penetrability of an object to the X-ray is in direct ratio to its specific gravity and density. When it is desired to establish the presence or absence of biliary or pancreatic calculi, no special preparations excepting the preliminary emptying of the bowel by the patient are necessary. Where, however, the organs are softer and less dense than the surrounding tissues, they do not show up in the X-ray. They may, however, be brought out by contrast, either by filling them with a still more penetrable medium such as air or gas (hydrogen, etc.), or with an opaque substance such as bismuth or barium. Inflation of the viscera by gas or air has been abandoned owing to its unsatisfactoriness. An opaque meal of bismuth or barium in liquid or pappy form conforms to the shape of the hollow organs, more or less completely filling them out, and by obstructing the X-ray, throws shadows on the fluoroscopic screen or photographic plate. The shadow represents, of course, only that part of the organ which is filled with the opaque meal and the density of the shadow is directly proportional to the amount of opaque material present. Thus, in a stomach with good tone, which contracts well upon its contents, the shadow indicates the true shape of the organ; while in an atonic stomach the food falls to the lowermost part and this alone shows in the shadow. If such a stomach is pressed upon, the shadow shows the upper part of the organ as well, but upon release of the pressure, again drops.

Any projection into the lumen, either from a growth in the wall or the pressure of a body from without, causes an indentation of the contour of the shadow, to which the name of "notch" or "shadow defect" has been given. A permanent shadow defect, evident upon repeated examination and change in the patient's posture, is quite characteristic of tumors in the wall of the organ. Most benign tumors, particularly submucous ones, are well circumscribed and have sharply defined borders; they therefore give a smooth shadow defect. But where they project into the lumen as polypoid tumors or have irregular surfaces as old indurated ulcers and broken down gummata, their shadow defects are irregular or mottled. Since malignant growths spread irregularly and their inner surface is not smooth, their shadow defects are irregular, uneven and ragged. This applies more particularly to the softer forms. The scirrhus variety, aside from its tendency to contract and produce constricting effects, is apt to infiltrate the walls and stiffen them, and thus cause a break in the peristalsis, and often a widening and gaping of the pyloric canal. Peristaltic breaks are best observed in fluoroscopy and shadow defects in the photographs.

Encroachments upon the lumen of the organs due to deepened peristaltic waves, spasms, scar or tumor contractions, angulations, kinks, etc., produce corresponding alterations of the bismuth shadow. To differentiate between a functional and an organic contraction, the use of various drugs such as atropin and papaverin has been suggested; but their action is unfortunately not always dependable. Better are massage, change of posture, or manipulation of the parts with the protected hand or a wooden spoon under the guidance of the fluoroscopic screen. Frequently these measures will cause a functional (spasmodic) contraction or incisura, as it is called, to disappear; if not, the ultimate test is to repeat the radiologic examination on different days. But even this last may fail to furnish the proper interpretation, as was experienced by the writer in a case of epigastric hernia which showed a large smooth defect on the greater curvature of the stomach, that persisted for days and was not removable through hand manipulations, etc. Exploratory laparotomy revealed no organic gastric lesion and radiological examination subsequent to the operative cure of the hernia, showed a normal greater curvature.

Any local or general enlargement of the lumen results in a corresponding increase in the shadow. This is seen in partial or complete obstructions, atonic dilatations, diverticuli and local bulgings due to surface adhesions or to the craters and niches of callous and penetrating ulcers. As regards these niches, a condition first described by Haudek, the extra shadow may be at some distance from the organ and usually persists even after the latter has become emptied.

The X-ray is our most valuable means of determining the size, form, position, fixity, tone, peristaltic activity and motility of these hollow organs. It is also of great use for testing the emptying power of these organs, or any portion thereof. It is our best procedure for ascertaining the local factor in constipation. The liver frequently shows up in large radiographs, indicating better than any other procedure its exact position. Tumors of the liver cannot be demonstrated, since their density is about equal to that of the organ itself. With specially developed technic, considerable advance has been recently made in the demonstration of the presence of gallstones. As is well known, their ease of demonstration is directly proportionate to their calcium content. Not infrequently, a diseased gallbladder is also seen in the radiograph. It has been stated that this only occurs where the bile or the walls of the gallbladder are thickened, but the writer has a plate showing a very distinct gallbladder in a patient in whom operation revealed normal conditions of that organ. That adhesions between the gallbladder and neighboring organs, such as the stomach or intestine, may be determined has long been known. Pancreatic stones, owing to their high calcium content, are comparatively easy to demonstrate with the X-ray.

Röntgenology has exposed the fallacy of the so-called high enema, proving that with a tube inserted only a few inches into the rectum and comparatively low pressure, the enema within a few minutes reaches up to and, in exceptional instances, beyond the ileocecal valve.

A negative finding with the X-ray may be of exceedingly great importance, enabling us in borderline cases to place the case into a medical or a surgical category. This applies, for instance, to that extensive class of cases occurring in middle-aged or elderly subjects in which there is a strong suspicion of beginning malignancy, that cannot be excluded or confirmed by the usual methods. Of this type are cases of recent esophageal obstruction, old gastric ulcers, and conditions associated with subacidity, achylia, anorexia, loss of weight, etc. In most instances dependence may be placed upon the radiographic findings, but just because of the exceptional cases, extreme caution must be exercised before accepting the röntgenological diagnosis as final, when it does not harmonize with the clinical facts.

Regarding the technic, it is not within the scope of this paper to discuss the many methods employed. For these the literature must be studied. The basic principles are definitely settled, but new phases are constantly being reported and there is as yet no uniformity as to the details of technic. This is but natural in an art that is of such recent development. An agreement can only be reached when the various plans of procedure have been thoroughly tested out and, by a comparison of the results, reliable standards evolved.

REFERENCES

Myer, Jesse W.—Jour. Am. Med. Ass., Nov. 29, 1913, p. ——

Basch, Seymour—Surg., Gynec. & Obstet., Feb., 1916, p. 165.

A very instructive discussion of the röntgenological diagnosis of carcinoma of the stomach is given by Carman, R. D., Am. Jour. Med. Sciences, Nov., 1915, p. 625.

Haudek—Muench. Med. Wochenschr., 1910, Nos. 30 and 47; ibid., 1911, No. 8.

THE DIAGNOSIS OF ABNORMALITIES OF MYOCARDIAL FUNCTION

BY T. STUART HART

Assistant Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University; Visiting Physician, Presbyterian Hospital
New York

VII

AURICULAR FIBRILLATION

The group which we are now to consider is characterized by a "complete irregularity" of the pulse. Waves of varying sizes, large and small, separated by intervals long or short, follow each other in a confused succession. The pulse is absolutely devoid of rhythm. The closest study will not enable us to predict whether the next event in the series is to be a forcible, or a weak impulse, a pause short or long. We may have a series of small waves separated by unequal intervals occasionally mingled with large waves, or large and small waves and longer or shorter pauses may be jumbled together in the utmost confusion.

In the older literature this pulse has been described and labeled with many different names, *"pulsus arhythmicus," "deficiens,"* *"inaequalis," "intermittens," "irregularis,"* it has been called the "mitral pulse." The pulse rate may be fast or slow, it may exceed 200, or may be under 40.

The heart shows the same degree of irregularity, indeed by auscultation the absolute irregularity is made more apparent than by palpating the radial. The heart activity is, I think, best described by the now discarded term *"delerium cordis."* The apex impulses are unevenly spaced, and forcible and weak thrusts are mixed in an erratic series.

The heart sounds show the same absence of rhythm both in time and force. They vary greatly in intensity. Each contraction may be represented by a first and second sound, or at more or less frequent intervals the first sound alone is audible suggesting that the feeble contraction has failed to open the aortic valves.

The credit of separating this type of disordered myocardial function from others belongs in a very large degree to Mackenzie* who pointed out the constant association of the "ventricular form of venous pulse" and "complete irregularity" of the ventricle. At this time he ascribed these phenomena to a paralysis of the auricles. Later he amplified his views in a paper based on the study of 500 cases.† In a series of subsequent papers he modified his theory of the underlying defective mechanism, and since he was led to believe that the auricle and ventricle contracted simultaneously, he assumed that a point near the *A-V* junction was the source of the impulse which simultaneously excited both the upper and the lower chambers. He further assumed that this abnormal pacemaker was located in the node of Tawara‡ and therefore introduced the term "nodal rhythm"§ to designate this group.

The real explanation of the activity of the completely irregular heart was made clear by the electro-cardiographic studies of Rothberger and Winterberg¶ and of Thomas Lewis.|| To these investi-

---

*Study of the Pulse, London, 1902.

†Amer. Jour. Med. Sci., 1907, CXXXIV, 12.

‡Quart. Jour. of Med., 1907-8, I, 39.

§With our present clearer insight into the mechanism of the completely irregular pulse, the term "nodal rhythm" should not be used in this connection. It should be reserved to designate a rare but definite cardiac activity in which the pacemaker is located in the *A-V* junctional tissues.

¶Wien. klin. Wochenschr., 1909, XXII, 839.

||Heart, 1909-10, I, 306.

gators, working independently, is jointly due the distinction of conclusively demonstrating that in these cases there is no gross auricular contraction, but that the auricular wall is in a continuous state of fibrillation. To those who are interested in the facts upon which their conclusions are based a study of the original papers is commended.

EXPERIMENTAL PRODUCTION AND MECHANISM

When the auricle of an exposed heart is faradized the coördinated contractions of the chambers as a whole cease, there is no auricular systole, and the muscle wall assumes the relaxed condition of diastole, the muscle mass is, however, not at rest, it manifests a continuous activity which consists of fine irregular waves with here and there a sharper twitching movement. The movement has been likened to the appearance of the squirming of a bunch of worms. This is what is known as *fibrillation of the auricle.* It is similar in appearance to the fine fibrillary movements which are not infrequently seen in the tongue. When such a condition is produced experimentally it is accompanied by a complete irregularity of the ventricle, a venous pulse of the ventricular form and an electrocardiogram which shows a continuous series of small irregular deflections, which are met with in no other condition, and are undoubtedly due to the abnormal auricular activity.

Lewis studied 3 horses, each having complete irregularity, the electrocardiograms were similar to those obtained from men with complete irregularity. On quickly opening the chest the auricles could be seen in a condition of fibrillation, and the ventricles were contracting in the characteristic irregular manner. Cushney and Edmunds,* who were familiar with the experimental production of auricular fibrillation, in discussing a case of complete ventricular irregularity in man were the first to suggest that this phenomenon might be associated with a fibrillating auricle.

Auricular fibrillation has been produced experimentally by increasing the intra-auricular pressure (Lewis), by stimulating the vagus and accelerator nerves (Morat and Petzetakis) by the application of heat to the myocardium (Lagendorff) and by toxic doses of drugs, digitalis (Francois-Frank), nicotine (Pezzi and Clerc), pilocarpine (Busquet).

---

*Amer. Jour. Med. Sci., 1907, CXXXIII, 67.

It has been suggested by Lewis* that fibrillation is due to a highly irritable condition of many points in the auricular musculature, each of which "is independently and spontaneously elaborating heterogenetic impulses," that is to say he views the condition as closely allied to the auricular extrasystole, but instead of there being one irritable point there are many, and the multiple impulses thus set free neutralize or reinforce one another in an utterly haphazard fashion.

Recently Garrey† has taken exception to this view, and substitutes the theory that the condition is due to many small blocks between the fibers of the auricular musculature, so that the fibers receive their stimuli not by the usual paths, but in such a manner that the stimuli pass slowly from cell to cell by a circuitous path, thus causing contraction waves in a shifting series of ring-like undulations.

There is little question that the ventricular contractions are the result of irregular impulses received from the auricle for the electrocardiographic ventricular complexes are usually of the form which are secured from stimuli arising in a point above the level of the junctional tissues. There are several factors which may have an influence in determining the instant at which the ventricle shall respond: (a) it is conceivable that a stimulus is effective only when of a certain magnitude, and that such a size is only attained when a considerable number of minute auricular impulses bombard the junctional tissues at the same instant, (b) this constant shower of auricular stimuli may have a modifying effect on the excitability or the conductivity of the bundle, (c) the underlying disease which has caused the damage to the auricular musculature may not have left the *A-V* bundle unscathed.

The peculiar susceptibility of cases of auricular fibrillation to stimulation‡ of the left vagus and the drugs of the digitalis group suggests a distinct change in the functional activity of the junctional tissues.

The force of the successive ventricular contractions depend mainly on the period of rest which precedes any particular contraction, thus permitting a more complete filling of the ventricle and a recovery of its contractile power, but as has been pointed out by Einthoven and Kortweg* this relationship is not always maintained, hence other factors must be at work which are not completely understood.

---

*Mechanism of the Heart Beat, London, 1911, p. 192.

†Am. Jour. Physiol., 1914, XXXIII, 397.

‡Draper and Robinson, Jour. Exp. Medicine, 1911, XIV, 217.

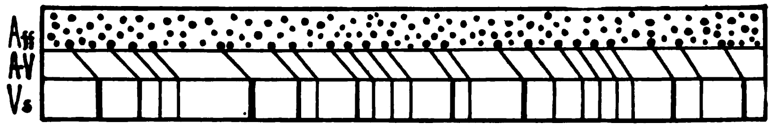


FIG. 1

Diagram to illustrate our conception of the mechanism of auricular fibrillation. Aff dots of various sizes indicate stimuli of various magnitudes arising in many parts of the auricular wall neutralizing or reinforcing or blocked off from one another. When effective the impulse is transmitted through the junctional tissues (*A-V*) and the ventricle responds (*Vs*) at irregular intervals with a varying degree of force, roughly proportional to the length of the preceding diastolic period. It is to be noted that the auricle is in a state of continuous activity, but there is no coördinated contraction: also that the stimuli which call forth a ventricular contraction arises in a point above the ventricular tissues.

The accompanying diagram (figure 1) portrays in a graphic manner the conception of the mechanism, the upper reaches of the junctional tissues are being continuously bombarded by a shower of small impulses from the auricular segments, at irregular intervals these stimuli become effective and a ventricular activity, irregular in time and force, is the result.

PATHOLOGY

Every heart showing fibrillation of the auricles which has been exhaustively examined has given evidence of gross or histological damage of its tissues, but as yet no lesion has been described to which we may definitely attribute the abnormal functional activity. Grossly one finds valvular defects, most frequently mitral stenosis, hypertrophy and dilatation, pericarditis and coronary sclerosis. Mackenzie early in his studies pointed out the frequency with which dilatation and hypertrophy of the auricles is met with in these cases. Histological examination almost invariably reveals evidence of acute or chronic inflammatory changes of the myocardium, leucocytic infiltration, or fibrosis and atrophy of the muscle cells. In several instances the blood supply of parts of the auricular wall has been found deficient. In some cases structural changes have been de-

*Heart, 1915, VI, 107.

scribed in the sinus node or in the *A-V* node, in others these tissues have revealed nothing abnormal to the most thorough search.*

The recognized fact that fibrillation of the auricles may be only temporary suggest that in a certain number of cases, the cause may be a toxin, or a temporary nutritional disturbance.

Since it has been shown experimentally that over distension of the auricle may lead to fibrillation, it seems reasonable to suppose that an auricular wall damaged by disease, under the stress of a defective valve, or the demands of a general arteriosclerosis, may readily fall into fibrillation. This would explain the onset in a large number of the cases that are seen in the clinic.

ETIOLOGY

Auricular fibrillation is met with in all decades of life, but is extremely rare in those under ten years of age. In a personal experience with over 300 cases I have seen only one case under the age of ten. A curve of the age incidence (figure 2) indicates that 89 per cent. of the cases occur between the ages of 21 and 60. The first sharp elevation occurs during the years when rheumatism is preva-

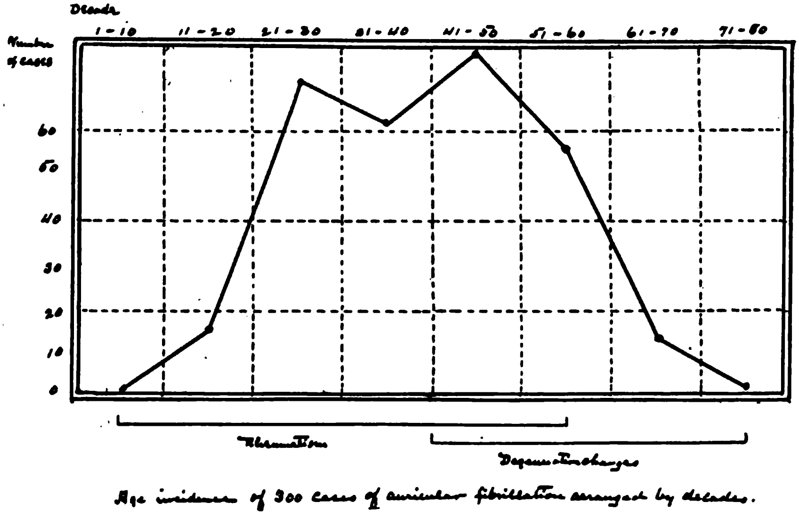


FIG. 2

Age incidence of 300 cases of auricular fibrillation arranged by decades.

---

*Cohn: Heart, 1912-13, IV, 221.

lent, the second elevation occurs in the decade when the rheumatic period overlaps the time at which arteriosclerotic changes become a prominent feature.

It is seen more frequently in men than in women, the proportion being about 2 to 1.

The association with mitral disease is very noticeable, 60 per cent. of the cases showed a mitral stenosis, with or without mitral incompetence, in 5 per cent. there was evidence of mitral insufficiency without stenosis. In a few cases there was an aortic lesion as well as a mitral defect. In only 7 instances have we seen fibrillation in hearts with defects of the aortic valves only.

Distinct evidence of one or more attacks of *acute rheumatic fever* was obtained in over one-half of the cases studied. The well-known frequency with which rheumatism affects the mitral valve is further evidence that this disease is the underlying cause of the myocardial defect. It is not, however, common to see auricular fibrillation during the first attack of rheumatic fever, the damaged heart does not usually go into fibrillation until some years after the evidence of a valvular defect has been established. This phase is further emphasized when we compare the age incidence of rheumatism and of fibrillation; according to Church* 57 per cent. of the mitral attacks of acute rheumatism occur under the age of 20. Our chart of the age incidence of fibrillation shows that in only 16 cases† did the onset occur before the 20th year, and only 149 cases‡ showed fibrillation during the first four decades. This suggests that fibrillation points to a rather advanced degree of tissue damage. The stretching of the injured auricular muscle under the stress caused by a defective valve may well be an important factor in producing this type of abnormal function.

We have seen fibrillation develop during the course of a lobar pneumonia on six occasions.§

In several cases with mitral disease the original lesion could be attributed to an attack of scarlet fever.

Attacks of influenza seem to be the only etiological factor that could be obtained in some of the patients. The most careful scrutiny of a case now under observation has given no explanation of a well marked mitral stenosis and auricular fibrillation other than repeated attacks of grippe.

---

*System of Medicine: Allbutt and Rolleston; Vol. II, Pt. 1, 603.

†8 per cent. of those having a rheumatic etiology.

‡77 per cent. of those of rheumatic origin.

§In 126 cases of pneumonia carefully studied by Dr. A. E. Cohn, fibrillation occurred in 12 cases (personal communication).

We have seen fibrillation of the auricles in four cases of Graves' disease, two of these gave an old history of attacks of acute rheumatic fever and each presented evidence of a mitral stenosis.

Aside from the rheumatic cases, and those in which the myocardial damage may be attributed to one of the acute infections, there is a considerable group which includes those suffering from degenerative changes, such as general arteriosclerosis, chronic nephritis, emphysema, etc. This group is composed of patients who, for the most part, have past their fortieth year, and in it males largely predominate.

Syphilis and alcohol appear to be prominent etiological factors in this group. Many of these have no discoverable evidence of valvular disease, a soft blowing systolic murmur in the mitral area is, however, not an uncommon feature. Several had dilatation of the arch of the aorta, and insufficiency of the aortic valves.

IDENTIFICATION

Clinical, palpation of the radial pulse in most instances is sufficient to permit us to assign this group of cases to their correct category. The complete irregularity is at once evident, the pulse beats are irregular both in time and force. As a rule the stronger beats follow the longer pauses, but the disorderly sequence of large and small waves separated by intervals long or short, allows us to make a very strong conjecture as to the kind of abnormal cardiac activity with which we have to deal. There are other cases in which the complete irregularity is not so easily detected by palpation. When the pulse volume is diminutive, when the pulse pressure is relatively small, the recognition of the unequal size of the successive waves may be quite difficult. In cases with a rate of over 160 the pulse waves may show very insignificant differences in size, and the time intervals may show variations only to be detected by the most careful measurements secured by instrumental means. There are certain patients with pulse rates between 70 and 80, and with the waves placed at such even intervals that an uncorroborated examination of the radial pulse is quite insufficient to classify it as one of complete irregularity.

Before going farther we should emphasize the point that while practically every case of "auricular fibrillation" is characterized by a pulse of "complete irregularity," there are other conditions which may also afford a "completely irregular" pulse which can only be differentiated by exact instrumental methods, while in nineteen out of twenty instances a "complete irregularity" of the pulse correctly indicates a condition of "auricular fibrillation," the twentieth may be the result of an entirely different heart activity. The types which may notably give rise to this confusion are certain "sinus arrhythmias," and cases with very frequent extrasystoles, particularly when they arise from several points in the heart wall, or are interspersed with short runs of paroxysmal tachycardia.

If the veins of the neck are prominent inspection will show that the jugular pulsations are synchronous with the apex beat, no presystolic wave can be seen, the venous pulse is of the ventricular form.

Auscultation of the apex will corroborate the complete irregularity discovered in the pulse, and at times will make clear an irregularity which was not discovered when palpating the wrist. The sounds vary greatly in intensity and follow one another at unequal intervals. Often the heart beats are far more numerous than the arterial pulsations, some contractions are too feeble to perceptibly increase the lumen of the radial artery, and others fail even to open the aortic valve. These latter are represented by a first heart sound only, the second sound is wanting.

The question as to whether the aortic valves are or are not opened by any particular ventricular contraction depends on the relative pressures in the aorta and the left ventricle. If the diastolic period is very short, the contractile power of the ventricle will have had little time in which to accumulate, at the same time the pressure in the aorta will be relatively high. Under such conditions the valves will not be opened. On the other hand given a longer pause the next succeeding ventricular contraction will have recovered its contractility to a greater degree, the aortic pressure will be relatively low, and the aortic valves will be opened.

In some cases where the degree of irregularity is moderate, it may be difficult to distinguish this from a condition of extrasystole. It is helpful in such cases while listening to the heart beats to concentrate the attention on the beats which occasionally occur too early, and which simulate the premature beat of the extrasystole, the diastolic period following these early contractions of complete irregularity usually do not have the same length, nor as a rule do they have as long a duration as that which would constitute the "compensatory pause" of an extrasystole, and thus may be differentiated.

If valvular defects are present murmurs are usually detected, but at times their character is quite different from those heard before the inception of the complete irregularity. If the heart is beating very rapidly the murmurs are less intense, and the sharp snap of the first sound, so characteristic of mitral stenosis, may become muffled. The intensity of the murmurs vary from cycle to cycle. The more forcible beats are accompanied by relatively loud and harsh murmurs, the more frequent feeble contractions by murmurs of less conspicuous intensity.

There is frequently an increase in the intensity and duration of the mitral diastolic murmur. It may also assume a rougher quality. This may be explained on the basis of the increased volume of blood under considerable pressure in the right auricle, which is possibly distended and is never emptied by an effective contraction.

For the most part, the time relations of the murmurs remain as before the change to complete irregularity, systolic and diastolic murmurs continue to occupy their established position in cardiac cycle. Mackenzie long ago pointed out one exception to this general rule, namely, the disappearance of an established presystolic murmur in cases of mitral stenosis which changed from a physiological rhythm to one of complete irregularity. His explanation of this phenomenon assumed that the presystolic murmur of mitral stenosis was due to auricular systole, and that the failure of the auricular contraction accounted for the disappearance of this murmur. While this phenomenon is frequently seen it is not invariable as has been evidenced both by the ordinary methods of eliciting physical signs* and by occasional graphic records of the heart sounds which may be found in the literature. Authorities are not at all agreed that this presystolic murmur is due to auricular systole, and the persistence of the murmur in certain cases of undoubted auricular fibrillation lends support to the view that other explanations of the mechanism of the production of this murmur may not be disregarded.

The character of the complete irregularity of the arterial waves are clearly brought out by taking a tracing of one of the peripheral arteries (radial, brachial or carotid), and by means of a simple graphic record of this kind (see figures 3, 4, 5, 6 and 7), one can hardly miss a correct diagnosis.

The great variation in the size and time intervals of the arterial pulse waves is shown in figures 3, 4 and 5. In figures 3 and 4 the waves are for the most part of large volume, but show considerable differences in force and time intervals. Figure 5, shows a brachial pulse rate of 138. Many of the smaller waves could not be detected by palpation of the radial artery, hence it is easily seen how a count of the radial alone would have been very misleading in determining the rate of the heart beat. Figures 6 and 7 are both taken from cases of auricular fibrillation. They indicate an unusual degree of rhythmicity, but careful measurements will show that the uniformity is apparent rather than real. In both of these records there are waves which a casual observer might mistake for extrasystoles, but close attention reveals not only a lack of uniformity in what one might take to be waves of the physiological rhythm, but further a period of very inconstant length both before and after the small waves which thus differentiate them from the intervals which one finds on either side of the extrasystole as commonly observed.

Polygrams make the diagnosis still more clear (see figures 8, 9, 10, 11 and 12). In these records the arterial curves show in considerable variety the features that have already been dwelt upon. In addition to this the jugular reveals features which are characteristic of this form of irregularity. It is itself completely irregular. The time intervals between the waves indicate a complete

---

*Hart, Med. Rec., New York, 1911, LXXX, 2.

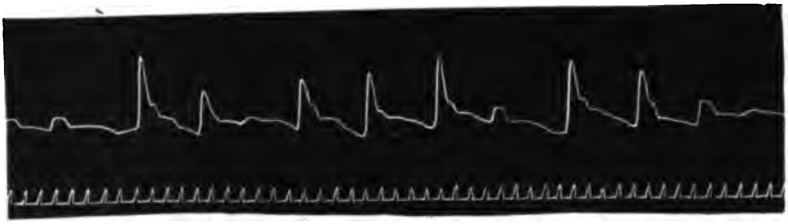


FIG. 3

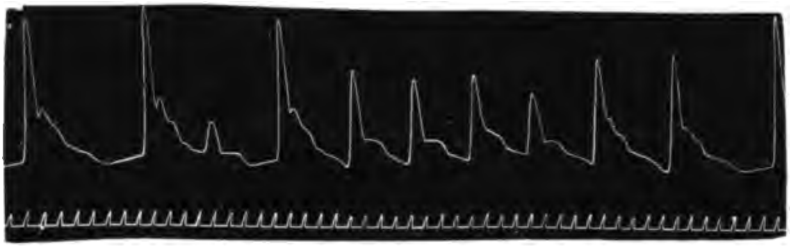


FIG. 4

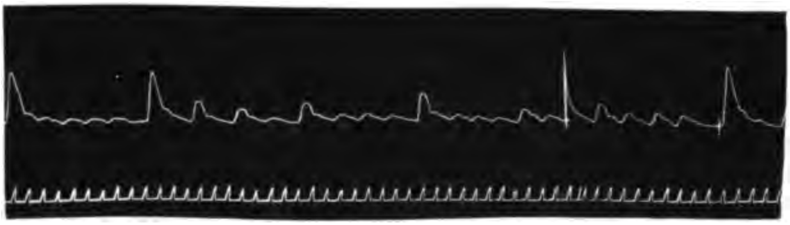


FIG. 5

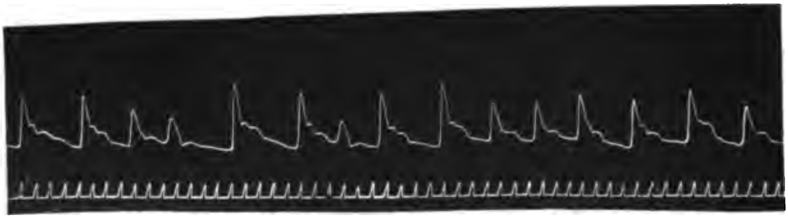


FIG. 6

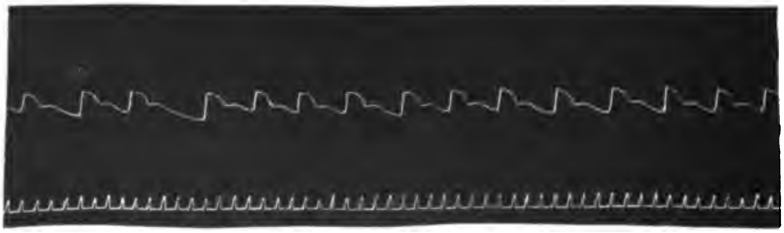


FIG. 7

Arterial tracings from cases of auricular fibrillation showing the great variety of pulses which are seen. All of these records show complete irregularity.

absence of rhythmicity. The size of the waves show great variations, which are quite independent of the phase of respiration in which they occur. Furthermore, the venous pulse is of the "ventricular form," that is to say all the positive waves occur during the ventricular systolic period, the *a* wave, the representative in the normal jugular of auricular systole is absent, *c* and *v* waves may be made out, the *c* wave usually varying in size proportional to the synchronous ventricular contraction. The *v* wave is usually a large broad elevation which frequently begins earlier than the *v* wave of the physiological rhythm and may be fused with the preceding *c* wave (figures 8, 11 and 12). The explanation of this phenomenon is found in the distended condition of the auricle which is probably never effectually emptied, hence the ventricular pressure is more readily transmitted by way of this blood column to the large veins. This feature may be further emphasized by a relatively insufficient tricuspid valve. The *v* wave is promptly terminated by the opening of the tricuspid valve, and the discharge of blood under abnormal pressure in the auricle into the ventricle. An examination of the several polygraphic records indicates the variety in the form and time of the beginning of the *v* waves. As a general rule the cases

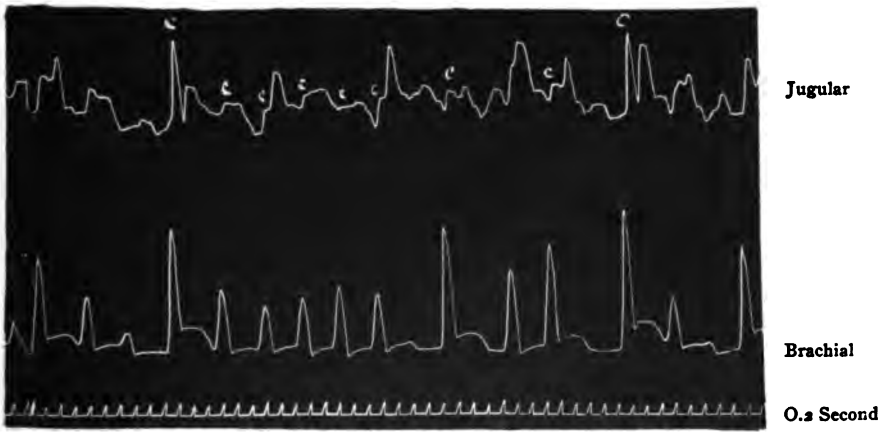


FIG. 8

Auricular fibrillation. Great variation in the size of the arterial waves and the intervals between them. Jugular completely irregular, *a* absent, *c* and *v* waves fused. Depression *x* absent.

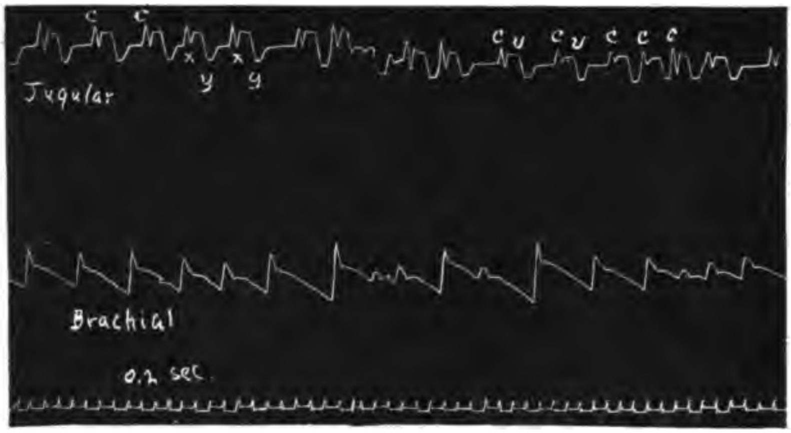


FIG. 9
Auricular fibrillation. At certain places the form of the arterial record might suggest an extrasystole. In the jugular record note the "entricular form" of the venous pulse, the *a* wave is absent.

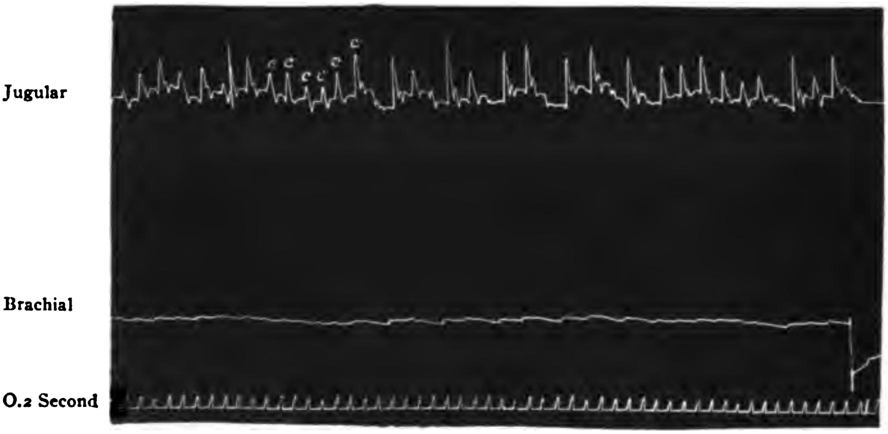


FIG. 10
Auricular fibrillation with a very rapid ventricular response. From a case of general arteriosclerosis with high blood pressure.

of auricular fibrillation of long standing with overdistended auricles and veins show a tendency for the *v* wave to become fused with the *c* wave, and the depression *x* may entirely disappear. The unusually deep depression *y* seen in figure 11, indicates an unusual fall of pressure when on the opening of the tricuspid valves the contents of the distended auricle are poured into a relaxed and dilated ventricle.

Figure 10 illustrates the kind of curves often secured from the cases of auricular fibrillation with a very rapid ventricular

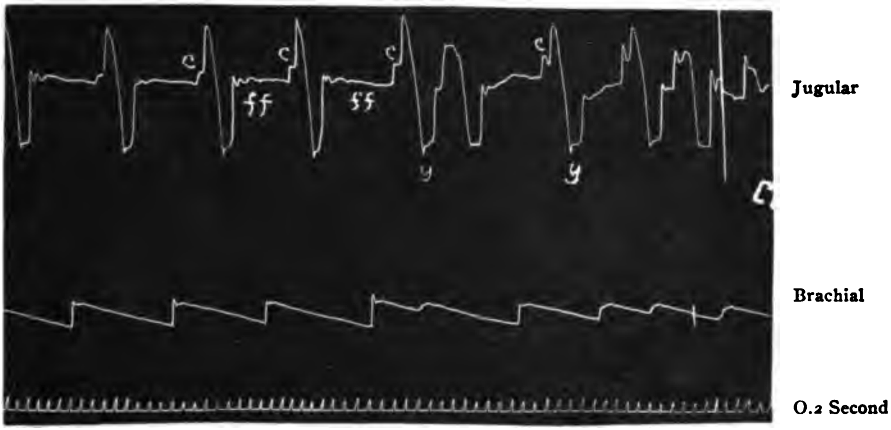


FIG. 11

Auricular fibrillation with slow ventricular response. Note absence of *a* wave, absence of depression *x*, unusual depth of *y*, small oscillations during diastole (ff).

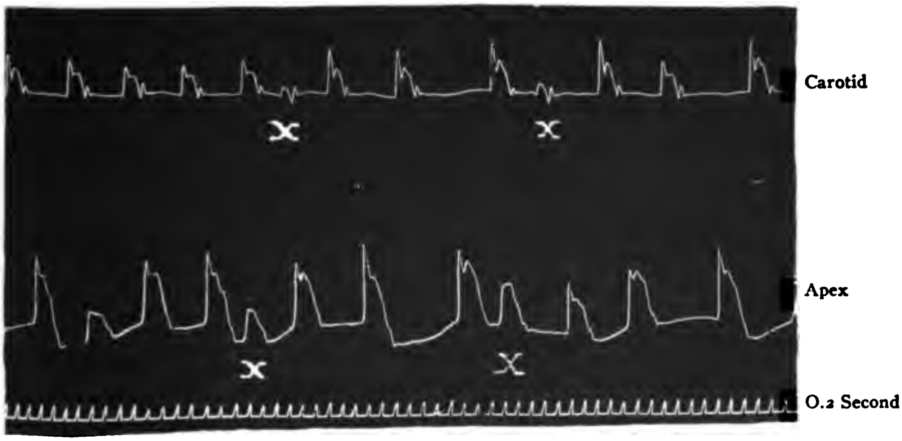


FIG. 12

Auricular fibrillation. Apex and carotid tracings. At X the weak ventricular contractions are barely able to open the aortic valves.

response in old arteriosclerotic cases, the pressure changes in the brachial and radial are often quite small, and it is difficult to obtain good arterial tracings.

A slow ventricular rate is recorded in figure 11. The brachial waves are well marked and extremely irregular at a rate of 60 per minute. In the jugular tracing there are seen during diastole some very small waves marked *ff*. These minute fluctuations in venous pressure are not infrequently obtained in auricular fibrillation when the ventricular response is deliberate, they have been ascribed to the undulating auricular activity, but it is quite probable that they are due to vibrations induced in the vein by the pressure of the cup used as a receiver.

Synchonous records of the apex beat and carotid are reproduced in figure 12. At *x* are seen weak ventricular contractions, which are barely forceful enough to overcome aortic pressure, and presumably would be too weak to be detected in the radial.

The *electrocardiogram* usually merely corroborates the evidence of auricular fibrillation obtained by the simpler methods of examination, but in certain obscure cases the electrical records are essential to a correct diagnosis. We have seen cases that have been carefully studied by skilful clinicians in which the irregularity was mistakenly interpreted as due to auricular fibrillation, and under vigorous treatment the patients grew worse rather than better. The galvanometric records disclosed the fact that these "complete irregularities" were not caused by "auricular fibrillation," and a proper revision of the treatment resulted in immediate benefit to the patient.

The distinctive features of the electrocardiagram of auricular fibrillation are:

1. Complete irregularity in the time intervals between the ventricular complexes.
2. An absence of the *P* wave.
3. A series of small waves which are continuous throughout the whole cardiac cycle.

If one examines a number of electrocardiograms from cases of auricular fibrillation such as are shown in figures 13, 14, 19, etc., the first thing that arrests our attention is the unequal spacing of the ventricular complexes. Some show this in a greater degree than others, but an absolute rhythmicity is almost* never seen. The parallelism between the irregularity of the ventricle and the arterial pulse is shown in figure 13 in which simultaneous curves of the galvanometer and the pressures from a cuff placed on the brachial are recorded.

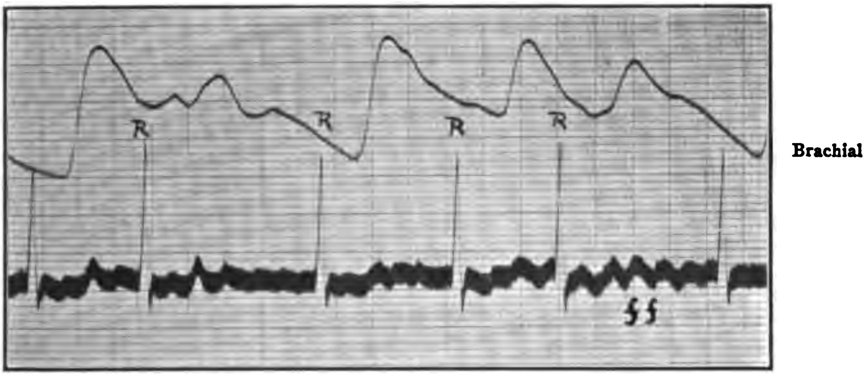


FIG. 13

Auricular fibrillation. Note the complete irregularity in both time and force of the brachial waves. The ventricular complexes of the electrocardiogram show the same irregularity, the *P* wave is absent, the small oscillations (*ff*) vary greatly in size and duration, but are present throughout the whole cardiac cycle.

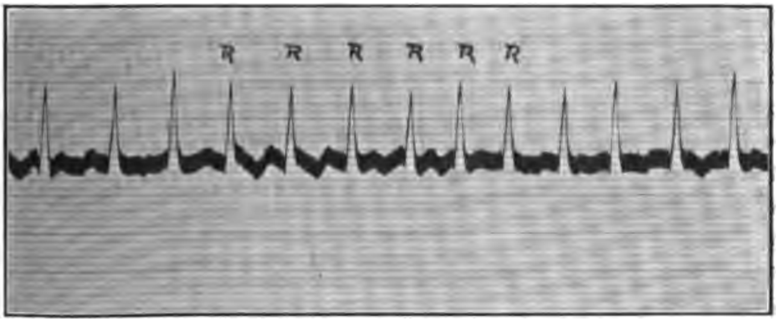


FIG. 14

Auricular fibrillation with rapid ventricular response. *R* varies in height according as it coincides with the summit of one of the small waves or a depression between two small waves.

---

*An exception to this general statement is seen in cases of auricular fibrillation complicated by an *A-V* block.

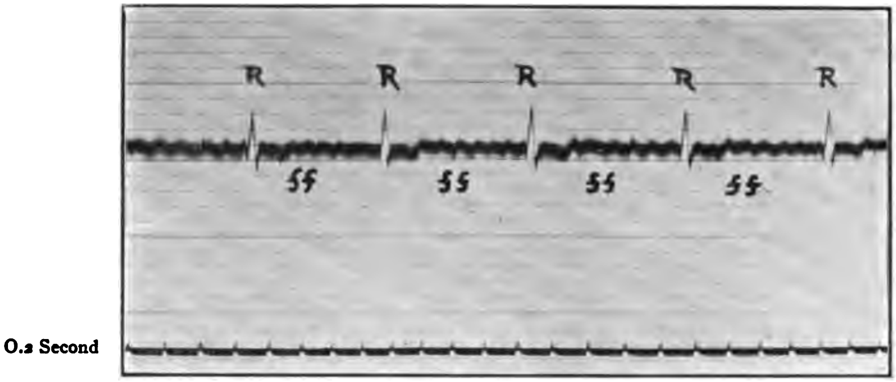


FIG. 15

Auricular fibrillation with ventricular response which is almost rhythmic. The auricular oscillations are small, but distinct, constant and quite uniform in size.

If we study the ventricular complexes carefully we are led to the conclusion that they have a normal form save for some distortion produced by the small waves of auricular activity which are present during the whole cardiac cycle, both systole and diastole. The ventricular complex may or may not be introduced by a *Q* deflection, both the ascending and descending limbs of the *R* wave are sharp and abrupt, *S* may or may not appear. *T* is usually present, the curve which is the result of the contraction of the lower chamber has the same characters and is of the same duration as is found in the same heart when the rhythm is of the physiological type. The records shown in figures 21 and 22 were taken from the same patient, figure 21 in June 1910 when the auricles were contracting efficiently. Figure 22 was taken 10 months later when the auricles were in fibrillation. A comparison of the ventricular portions of the two records show a marked similarity, the *R* and *T* deflections are of the same order, and of the same duration. We must therefore conclude that even when the auricles are in a state of fibrillation the ventricles receive their stimuli from a point above the bundle of His, and that the stimulus travels over the ventricle by a path that is normal in all respects. The ventricle responds to a supraventricular impulse.

The second feature of importance in establishing the nature of the cardiac activity from a study of these records is the absence of the *P* wave, the normal representative of gross auricular contraction. In certain records (figures 19, 23 and 27) one might at first glance question whether in some of the cycles a wave preceding the *R* deflections was not in reality a *P*, but an examination of a number of cycles will soon convince one that the inconstancy in the size, contour and time relation to the beginning of ventricular activity, make it necessary to find another interpretation.

In the electrocardiogram of the normal heart it may be recalled that in the period from *T* to *P*, the diastolic interval, there is a line without any suggestion of deflection. In the records now under discussion (figures 13, 14, 15, etc.) this period is evidently occupied by a continuous series of uneven oscillations. These are the representatives of the rapid, irregular fibrillary activity of the auricles. Further examination will convince one that these small deflections are not limited to the diastolic period, but are continued during systole as well, so that the whole curve is modified by an unending series of these small fluctuations. It is for this reason that in many of the records the ventricular complexes seem to be of abnormal form. They are merely distorted by the superimposition of these representatives of unceasing auricular activity. The *T* wave being relatively small is frequently very much changed in appearance. The *R* deflection when large is only slightly modified, but will fluctuate in height (figures 13, 14) according as it is coincident with a summit of, or a depression between the small waves. The oscillations are usually arrhythmic, and vary in frequency. When the rate can be estimated it will be found to be between 400 and 600 per minute: they are usually most conspicuous in records taken by the second and third leads.

The great variety in the rate, size and rhythm of the waves of fibrillary activity as shown in the electrocardiogram indicates great differences in the algebraic sum of the electrical potentials developed from moment to moment in the same auricle, and in different auricles. The oscillations may be small and fairly rhythmic throughout the whole record as seen in figure 15, or large and very arrhythmic (figure 16). Some curves show a great variation from cycle to cycle (figures 13 and 14), others small fluctuations which are inconspicuous but quite constant (figures 17 and 24). One may at times be confused by small rapid oscillations due to tremor of the skeletal muscles, when during the recording period the extremities are held tense and unrelaxed, these fluctuations are very fine and much more rapid than the waves of auricular fibrillation. They may be seen in figures 18, 19 and 20 taken from the same patient on successive days before, during and after a paroxysm of auricular fibrillation. These muscular tremors are met with in patients under excitement who make a voluntary effort to hold themselves quiet. If the rapidity of the oscillations are noted they should not be confounded with the waves of auricular fibrillation which have a much slower period of vibration.

In cases such as are shown in figures 15, 16 and 17 the arrythmia of the ventricles is only moderate, and it is plain that palpation of the radial or auscultation of the heart might fail to reveal the "complete irregularity." The graphic records, however, make clear the nature of the abnormal activity. The ventricle is seen to be definitely arrhythmic, *P* waves are absent, and the continuous series of oscillations, large or small, are seen during the whole cycle. In figure 17 a simultaneous jugular curve has been recorded. In this may be noted the absence of the *a* waves, the *c* and *v* waves participating in the ventricular

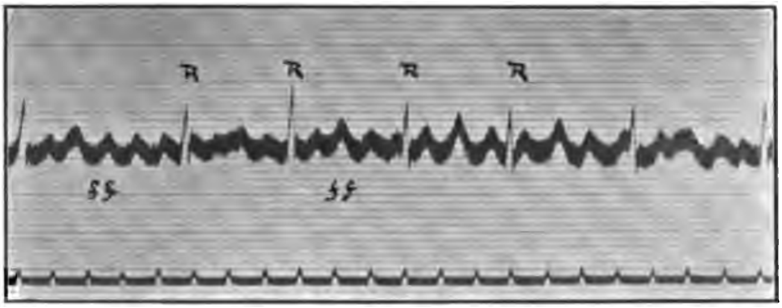


FIG. 16

Very large waves of auricular activity which distort the ventricular complex to a marked degree.

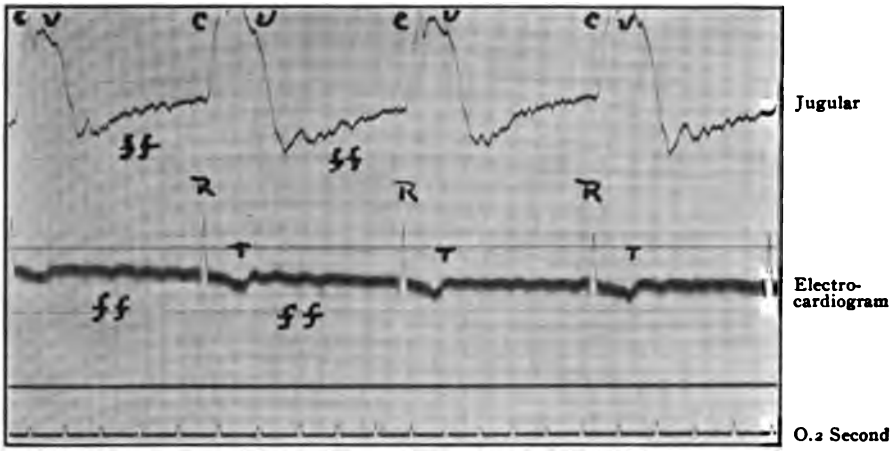


FIG. 17

From a case of auricular fibrillation with a slow ventricular response. The form of the ventricular complex indicates that the stimulus which called it forth originated in the supraventricular tissues. *P* is absent. *R* and *T* are but slightly distorted by the superimposed oscillations of auricular activity (*ff*). Upper curve obtained from the right jugular vein, *a* is absent, *c* and *v* have coalesced.

arrhythmia, and an absence of the depression *x* due to the early onset of the *v* wave, the fine oscillations *ff* are also apparent, but as has been pointed out in an earlier paragraph these are probably not characteristic of the fibrillating auricle, but are due to a venous thrill induced by the pressure of the cup used as a receiver.

CLINICAL FEATURES

Practically all patients with auricular fibrillation suffer from some degree of cardiac insufficiency. The functional disability of the heart may show a very wide range in different patients, and in the same patient at different times it may vary from a condition of almost complete heart failure to one in which there is ordinarily no evidence of an inability on the part of the heart to maintain an adequate circulation, and in which symptoms of insufficiency develop only under unusual stress. The degree of circulatory embarrassment depends in a very large measure on the integrity of the ventricular muscle. The valvular defect which so often has preceded the development of fibrillation has thrown upon the ventricles abnormal work, and perhaps injury which may, however, have been completely met by a compensatory hypertrophy. More often the underlying disease which has damaged both valves and auricular walls has also attacked the ventricles leaving them an easy prey to the stress which the inception of the new rhythm imposes. If therefore the ventricular myocardium is extensively damaged one may expect the rapid development of the classical symptoms of cardiac insufficiency, lowered arterial pressure, increased venous pressure, slow capillary flow, oedema, cyanosis, dyspnoea, congestion of the lungs, liver, kidneys, etc., etc. On the other hand with a reasonably healthy ventricular muscle the strain occasioned by the disorder in the upper chambers may be supported with comparatively little evidence of abnormal blood distribution.

Fibrillation once established usually persists to the end of life, hence the term *pulsus irregularis perpetuus,* which was formerly used to describe the arterial features. However, one sees cases from time to time in which the normal pacemaker reasserts its control, and a physiological rhythm is regained. This has occurred in 6 per cent. of the cases which have come under my observation. Such a case is shown in figures 18, 19 and 20 taken on successive days, April 19th, 20th and 21st, 1911. The records of the 19th and 21st, show a sequential rhythm, on the 20th (figure 19), the auricles were in fibrillation.

The patient is not necessarily conscious of the irregular heart action. At times they apply for examination on account of the sensation of "fluttering" or "thumping" in the precordial region or complain of "palpitation." More frequently dyspnoea or oedema are the symptoms which bring them to the physician, and the erratic and tumultuous heart activity has passed unnoticed. Very few of them complain of anginal pains, but tenderness in the precordial region is a symptom frequently elicited during the examination with the finger, or the bell of the stethoscope. In cases which have been watched over a long period, one will often see the following series of events: (1) Several attacks of rheumatic fever with arthritis and endocarditis, most frequently involving the mitral valve. (2) Later extrasystoles, often auricular in origin. (3) The heart becomes gradually dilated under physical stress, and the auri-

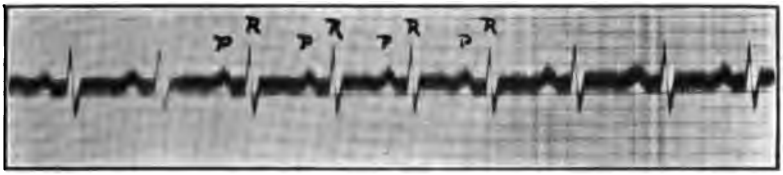


FIG. 18

The two following figures were obtained from the same patient on successive days, April 19, 1911. The rhythm is sequential. The heart is rhythmic, the auricle has its normal activity, as indicated by the regular appearance of the *P* deflection.

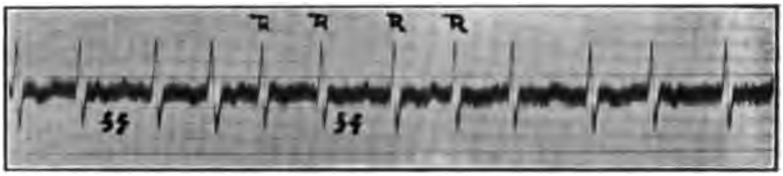


FIG. 19

Same patient as figures 18 and 20. Obtained April 20, 1911. At this time the auricle was temporarily fibrillating. Note the similarity in the form of the ventricular complexes in these three records. In this curve *P* is absent, there is complete irregularity, the small deflections (*ff*) due to the fibrillating auricle are present.

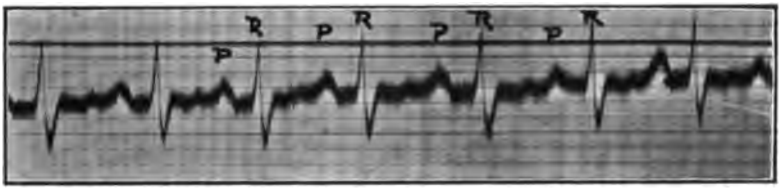


FIG. 20

Obtained from the same patient as figures 18 and 19, on April 21, 1911. The auricle has resumed coördinated contractions and the rhythm is again sequential. In all three of the above records note the fine rapid oscillations which are due to a tremor of the skeletal muscles.

cles begin to fibrillate, and usually continue this activity up to the time of death, which may be postponed for many years.

Such a sequence of events is shown in figures 21, 22 and 23. The patient, a man of 50, had a sharp attack of rheumatic fever followed by endocarditis when he was 20 years of age. At that time he was seen by Dr. E. G. Janeway, who told him he had a mitral stenosis. I first saw him in 1909 when he became conscious of an irregularity of the heart. At that time the rhythm was sequential, but he had

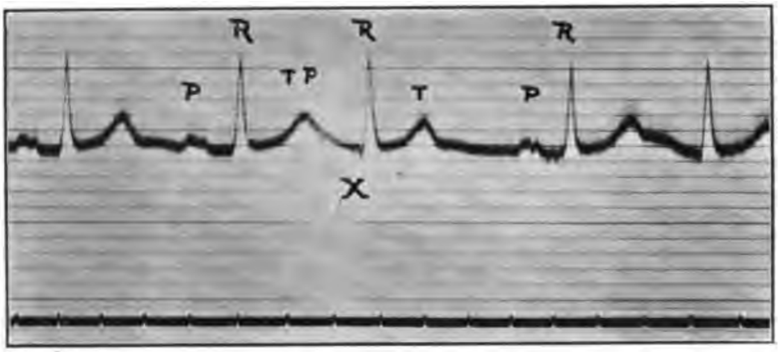


FIG. 21

Patient B. S., June 7, 1910. The auricle is contracting as a whole. *P* is broad and split. Extrasystole of auricular origin at X. Note fine tremor of skeletal muscles.

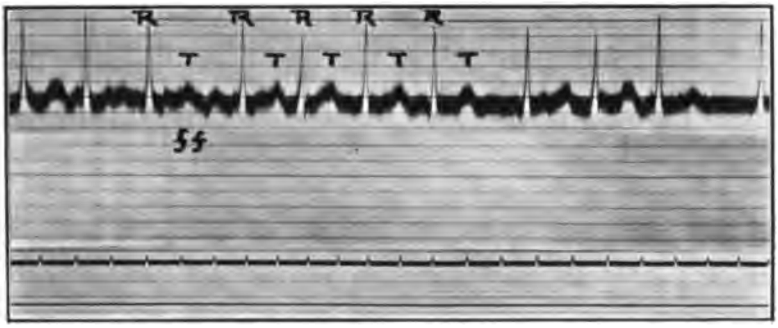


FIG. 22

Patient B. S., April 28, 1911. The auricles are now fibrillating, the rhythm is rapid and very irregular.

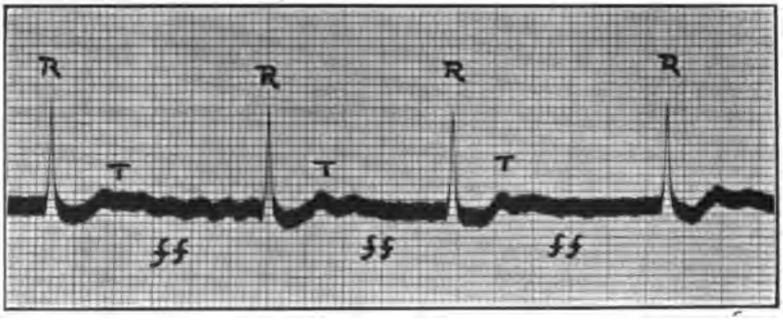


FIG. 23

Patient B. S., April 9, 1915. Figures 21, 22 and 23 are all from the same subject. The auricles are still fibrillating, but the ventricular response is much less rapid and more evenly spaced.

occasional extrasystoles, which were verified by polygraphic tracings. On June 7, 1910, the curve shown in figure 21 was obtained. It shows an extrasystole of auricular origin, and a split *P* wave. During an attack of pneumonia in February, 1911, his auricles began to fibrillate. Figure 22 was obtained on April 28, 1911, and figure 23 secured April 9, 1915. This has continued up to the present time (March, 1916). He is now in excellent health, and is able to conduct a business involving large responsibility but slight physical exertion.

The rate of the heart beat in auricular fibrillation is extremely variable. One sees cases in which the rate does not exceed 40 a minute, and others which exceed 200. Under appropriate treatment it is not unusual to see a rate of 140 reduced to 100 in a very short time.

The palpation of the radial pulse is a very insufficient criterion of the condition of the circulation. While the palpation of the pulse alone may be sufficient to establish a diagnosis, and while by this method one immediately detects the complete irregularity in force and frequency which characterize this group, the count of the radial pulse may be misleading. Frequently the number of the impulses which can be counted at the wrist is far below the actual number of cardiac contractions. Only those waves which are of considerable volume and force can be felt at the wrist (see figure 24), and many small ventricular contractions expend their force before reaching the radial, and some even fail to open the aortic valves. These small contractions are ineffectual in maintaining an adequate circulation, yet are exhausting to the heart muscle, for we know that, in accordance with the law discovered by Bowditch, every contraction of heart muscle is maximal, that is to say, if it contracts at all, it exhausts all of the energy stored as contractile material in its muscle fibers at any particular moment; hence it is evident that however small a contraction may be, it must be taken into consideration in estimating the gravity of the condition of any particular heart.

The inadequacy of the observations on the radial pulse alone is well illustrated in figure 25. Here the lower margin of the shaded area indicates the radial count, if one were guided by this alone, one would have said that on admission the cardiac rate was under 70 and never above 100. The upper boundary of the shaded area is the count taken by auscultation at the apex, and represents much more accurately the true condition, the admission rate being 127;

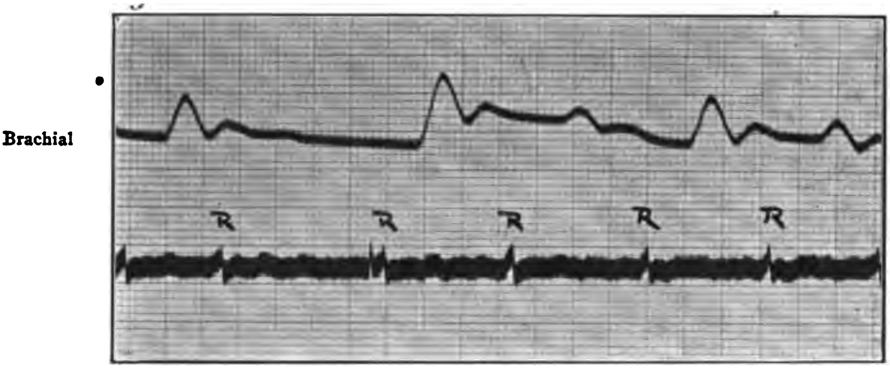


FIG. 24

Auricular fibrillation. Simultaneous records of the cardiogram and the brachial pulse. Showing the mechanism of the "pulse deficit." Every other heart beat has little or no effect in raising the pressure in the brachial artery.

the gradual reduction to the neighborhood of 60 makes the real improvement apparent.

The term *"pulsus deficiens"* has for a long time been used in describing pulse phenomena (Traube, Hering, Wenckebach, etc.),

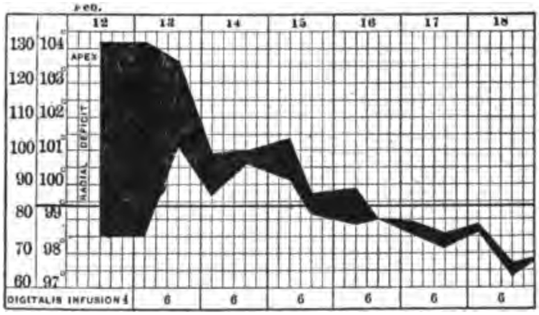


FIG. 25

The shaded area represents the pulse deficit; the upper edge is the apex rate; the lower edge is the radial rate. Figures in digitalis column indicate dosage of the infusion in drams per twenty-four hours. Patient in bed during period represented in the figure.

but each author has used it with a different meaning; some have considered it synonymous with *"pulsus intermittens,"* others have applied it to an absence of ventricular contraction, which breaks the ordinary rhythm; it has been used in describing extrasystoles and pulse alternans.

By *"pulse deficit"* we mean the difference between the number of cardiac contractions and the number of impulses which can be palpated in the radial artery. The best way of determining the pulse deficit is to have the apex counted by auscultation by one observer, while another is simultaneously counting the radial (these observations must cover a period of not less than a full minute, on account of the extreme irregularity of the pulse in many of these cases a count of one-quarter or one-half minute only is much less accurate). When one is obliged to make these observations unaided, the apex and the radial counts may be made in successive minutes. This of course does not give an absolutely accurate deficit, but it is extraordinary how closely the counts of successive minutes will coincide even when the heart and radial show the most extreme degrees of irregularity. After a little practice one may be able simultaneously to auscultate the apex and palpate the radial, thus determining the number of beats which fail to reach the wrist in the period of a minute.

In the figures the upper margin of the shaded area represents the apex count, the lower margin the radial count, and the width of the shaded area represents the deficit at any particular point in the curve.

The relative deficit. In many cases of auricular fibrillation, particularly where improvement has occurred and the heart has become less irregular, slow, and fairly compensated, it will be found that the count at the apex and the radial are identical. Even in these cases the individual waves show a considerable variation in force and size. This is brought more clearly to view if the cuff of a blood-pressure apparatus is placed on the arm and the radial is counted while varying degrees of pressure are applied through the cuff. This difference in the pressure values of successive waves we have termed the "relative deficit," as contrasted with the absolute deficit, when without brachial pressure some waves fail to reach the radial.

The following observation will serve to illustrate this point: The patient was a gentleman who had fibrillating auricles for something over two years, with at times an apex rate of 160 and an absolute deficit of over 50. Later his heart was fairly compensated, and he was able to supervise large business interests which required a daily attendance at his office of six to eight hours. When we last saw him his apex rate was 64, radial rate 64, deficit 0. On applying the brachial cuff the following counts were obtained:

Brachial pressure	Radial count per minute
140 mm. Hg.	0
130 mm.	50
120 mm.	58
110 mm.	62
100 mm.	64

While he had no absolute deficit, his relative deficit was quite evident when the pressure values of the waves of one minute were thus studied. This relative deficit may be detected in all cases of auricular fibrillation; it is rarely seen in other cardiac arrhythmias. We have found that such observations on the relative deficit are of real diagnostic value in corroborating a condition of auricular fibrillation which palpation and auscultation have led us to suspect.

The usual way of estimating blood pressure is entirely fallacious in auricular fibrillation. The accustomed method of obliterating the brachial artery by cuff-pressure and then by gradually lowering the pressure to determine the systolic blood-pressure by the height of the mercury column at which the pulse wave below the cuff is detected by palpation or auscultation is obviously of little value when practically each pulse wave has a different pressure value.

If such a pulse is observed for a period of a minute it will be found that only a small fraction of the total number of cardiac contratcions have a pressure value, approximating the systolic blood-pressure as determined by this method. Figure 26 illustrates the inaccuracy of this method in these cases. Systolic blood-pressure taken by the usual method would signify that the successive blood-pressures of this patient were 165, 160, 160, 162, 150, 155, 148, etc., and that as her condition improved the blood-pressure was lowered. As a matter of fact, only a few beats could be detected below the cuff when exerting a pressure at these levels, and while doubtless her systolic blood-pressure was momentarily at these levels, they in no way indicate the efficient pressure of the blood column. As we shall show later, her systolic blood-prssure really increased with the improvement in her condition.

A much more valuable estimate of the force of the blood-stream can be obtained by estimating the blood-pressure by another method; *the average systolic blood-pressure.*

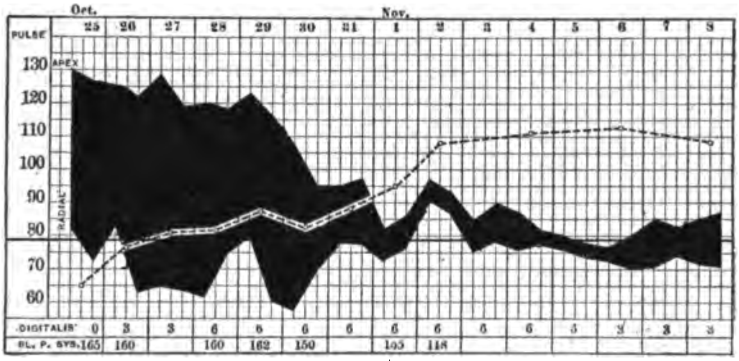


FIG. 26

The shaded area represents the pulse deficit; the upper edge is the apex rate, the lower edge the radial rate. The broken line indicates the "average systolic blood-pressure" (compare these values with the figures at the bottom of the chart which show the systolic blood pressure determined by the usual method). Figures in digitalis column indicate drams of the infusion per twenty-four hours.

To obtain what for convenience we have termed "the average systolic blood-pressure," the apex and radial are counted for one minute, then a blood-pressure cuff is applied to the arm, and the pressure raised until the radial pulse is completely obliterated; the pressure is then lowered 10 mm., and held at this point for one minute, while the radial pulse is counted; the pressure is again lowered 10 mm., and a second radial count is made; this count is repeated at intervals of 10 mm. lowered pressure until the cuff-pressure is insufficient to cut off any of the radial waves (between each estimation the pressure on the arm should be lowered to zero). From the figures thus obtained the average systolic blood-pressure is calculated by multiplying the number of radial beats by the pressures under which they came through, adding together these products and dividing their sum by the number of apex-beats per minute, the resulting figure is what we have called the "average systolic blood-pressure." The following two observations made on a patient will indicate the method of computation:

B.S., April 29, 1910. Apex, 131; radial, 101; deficit, 30.

Brachial pressure.	Radial count.
100 mm. Hg.	0
90 mm.	13 $\quad 13 \times 90 = 1170$
80 mm.	$47 - 13 = 34 \times 80 = 2720$
70 mm.	$75 - 47 = 28 \times 70 = 1960$
60 mm.	$82 - 75 = 7 \times 60 = 420$
50 mm.	$101 - 82 = 19 \times 50 = 950$

Apex = 131)7220

Average systolic blood pressure 55 plus

B.S., May 11, 1910. Apex, 79; radial, 72; deficit, 7.

Brachial pressure.	Radial count.
120 mm. Hg.	0
110 mm.	44 $\quad 44 \times 110 = 4840$
100 mm.	$64 - 44 = 20 \times 100 = 2000$
90 mm.	$72 - 64 = 8 \times 90 = 720$

Apex = 79)7560

Average systolic blood pressure 95 plus

The estimation of blood-pressure by this method gives us a simple and approximate measure of this factor of the heart's work. The diastolic pressure may be roughly determined by taking a graphic record with the Erlanger or Uskoff instruments and noting the pressure at which the average excursion of the pulse waves is maximal.

Examined by this method, the two groups, rheumatic and arteriosclerotic, show very different features.

In the rheumatic-mitral stenosis group, one not infrequently sees an average systolic blood-pressure under 70 mm. of mercury; these are usually cases with rapid rates (over 140) and a marked degree of cardiac insufficiency. With improvement the blood-pressure rises and may reach 120 mm., rarely 140.

In the arteriosclerotic group with fair compensation the average systolic blood-pressure is usually 160 mm. or over, and only falls below this when insufficiency becomes evident; in this group when the pressure, estimated in the above manner, falls below 140 mm. myocardial failure is very threatening.

The irregular activity of the ventricle is unquestionably the result of the peculiar activity of the auricles; while the upper chambers exhibit no gross contractions their walls show an incessant activity composed of irregular incoördinated contractions of the muscle fibers; impulses from the auricles are showered upon the junctional tissues in an entirely haphazard fashion, and the ventricles respond in utter confusion devoid of rhythm, and with extreme variation in the force of the succession beats.

In certain cases the junctional tissues between auricles and ventricles is somewhat damaged so that conduction between the chambers is depressed and fewer auricular impulses can reach the ventricles (figure 15); under these conditions the ventricular rate is not excessive, and the contractions show more uniformity in force. Here one is impressed with the small amount of disability which results from the fibrillating auricle; it is little more than a venous reservoir, and takes little part in moving the blood, and if the irregular impulses which are initiated in its wall do not disturb the ventricle too greatly, the circulation is maintained with a reasonable degree of efficiency. The fibrillary activity may be continued for years, and in itself is not at all incompatible with life or a proper distribution of the blood to the various organs.

PROGNOSIS

This depends largely on two factors; first the integrity of the ventricular muscle, and second the facility with which the shower of impulses coming from the auricle can be blocked. With a weak, dilated ventricle showing irritability as evidenced by frequent ventricular extrasystoles arising from points in the ventricular tissue other than the *A-V* bundle, the outlook is bad, and yet under skillful treatment and favorable conditions, such a heart may occasionally recover a considerable degree of efficiency, and life may be prolonged several years. Mere dilatation of the ventricle if otherwise healthy need cause us much less concern if careful treatment is instituted soon after the beginning of the complete irregularity; but the longer the period between the onset of fibrillation and the employment of correct therapeutic measures, the more difficult is it to restore the damaged ventricle. In the majority of instances digitalis will effectually block the erratic auricular impulses, and give the ventricle the lengthened diastolic period so necessary for its recuperation. In a few cases digitalis either cannot be taken in sufficient amounts, or fails to obstruct the stimuli from the upper chamber. In these the immediate prospect is exceedingly alarming.

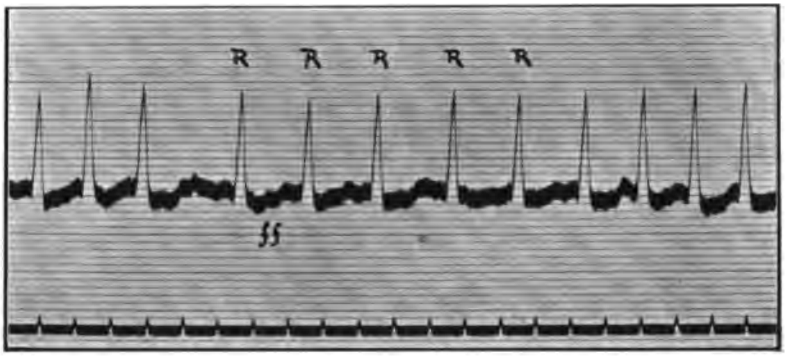


FIG. 27
Auricular fibrillation. Patient C. F. Heart very rapid and irregular (see figure 28).

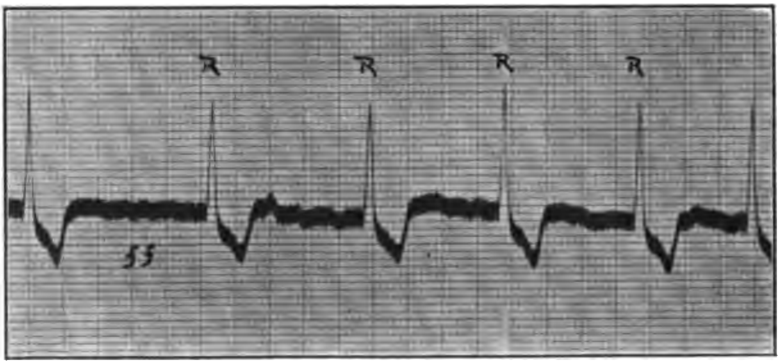


FIG. 28
Auricular fibrillation, patient C. F., taken one week after figure 27. The auricles are still fibrillating, but the heart has become slow under appropriate treatment. This is a favorable response to therapeutic measures, hence in this case the prognosis is good.

The degree of the response of the heart to treatment (see figures 27 and 28), and the amount of reserve force which can be secured for it, are our best indications of what the future holds in store. Many of these hearts may be brought to a fair degree of efficiency, but the reserve force is never very great, and neglect of treatment, or over exertion will almost invariably entail an attack of cardiac insufficiency. Each insult of this kind is met with greater difficulty and with each attack the outlook becomes more grave. Auricular fibrillation indicates a very serious myocardial defect; heart failure may be postponed for many years, but the majority succumb within ten years of its onset.

The signs which point to a favorable prognosis are:—

1. The resumption of a physiological rhythm.
2. The maintenance of a rate under 70.
3. The absence of a pulse deficit.
4. The absence of extrasystoles.
5. An average systolic blood-pressure of over 110 in the rheumatic group, and of over 160 in the arteriosclerotic group.

The symptoms which make the outlook grave are:—

1. A ventricular rate remaining for more than a few days above 130.
2. A persistent pulse deficit of 20 or over.
3. The occurrence of frequent ventricular extrasystoles.
4. A falling average systolic blood-pressure.
5. A ventricular rate which shows wide fluctuations under slight physical or emotional stress.

The gravity of a given case is often indicated by the ***amount of treatment*** requisite to secure a slow ventricular rate without a pulse deficit; some cases require very little treatment, and in these the immediate prognosis is good; others yield only to the most active therapeutic measures applied over a very long period. In such the danger is about proportional to the therapeutic measures found necessary.

DIAGNOSTIC ELEMENTS OF PONTO-CEREBELLAR TUMORS

BY ALFRED GORDON

(From the Neurological Department of the Douglass Memorial Hospital)
Philadelphia

Neoplasms situated in the angle formed by the cerebellum and pons present symptoms referable to the cerebellum, to the tracts in the pons and to the cranial nerves which emerge in close proximity. The diagnosis as a rule is not difficult. Nevertheless in some cases we hesitate. If the tumor grows from the cerebellum, cerebellar manifestations will appear first. If the tumor originates in the cranial nerves, the eighth most frequently, symptoms of the latter will be observed at the beginning. In the case reported below for several months the clinical picture consisted only of general manifestations of intracranial pressure, such as headache, vomiting and vertigo, but there were practically no localizing symptoms. The latter developed very slowly and they were at first so slight that they could be easily overlooked. While at autopsy the eighth nerve was markedly involved, viz., thickened and adherent to the neoplasm, nevertheless the impairment of hearing appeared late in the disease, only shortly before the patient died. The symptoms were so slight in the beginning that another affection than that of the cerebello-pontine angle was thought of. However, very close examination with special attention directed towards the minute details as to cranial nerves, to disturbances of synergic action of the upper and lower limbs, to the state of reflexes, to the gait and station—an examination of this type will invariably reveal a predominance of symptoms on one or the other side. This predominance may be, it is true, small and even very small, nevertheless if on repeated examinations it is found, the diagnosis will be greatly facilitated.

The investigation should first of all be directed to the cranial nerves. As the eighth, seventh, sixth and fifth nerves are most frequently involved, the following symptoms will be observed: Anesthesia of certain portions of the face on the side of the tumor including the cornea (fifth nerve); facial paralysis of the peripheral type with all the characteristic symptoms (seventh nerve); paresis or paralysis of the external rectus muscle of the eye on the affected side, hence internal strabismus (sixth nerve); impairment of hearing on the side of the tumor, also vertigo with a tendency to fall to the affected side,—all conditions caused by involvement of both branches of the acustic nerve.

Next in diagnostic importance stand the manifestations referable to the cerebellum with its peduncles. They are: (a) staggering gait which may be toward one or the other side, but invariably to the same side; the persistent unilateral sway in walking or standing is highly suggestive of a cerebellar involvement and especially of one of its lateral peduncles. (b) Ataxia of one of the upper or of the lower extremities is another important cerebellar symptom. It can be tested by the finger to nose movement and then we observe not only incoördinate movements but also overaction, dysmetria or hypermetria, the finger in trying to find the nose will more or less considerably overstep the point of destination. The same may be observed in flexing or extending various segments of the limbs. (c) To test the lower extremities for coördinate movements the patient is told to place the heel of one foot on various parts of the other lower limb, the same overaction may then be observed. (d) Adiadochokinesia is also a phenomenon characteristic of cerebellar lesions. It consists of an inability to rapidly pronate and supinate the hands. If in comparing these movements in both hands it is found that one of them is lagging and cannot keep pace with the other, a unilateral involvement of the cerebellum is to be suspected, the affected hand corresponds to the side of the lesion. (e) The position of the head in cases of cerebellar involvement has been considered as a localizing sign. While not all authors agree upon this point, nevertheless the head was found sufficiently frequently inclined towards the side of the tumor to be considered of diagnostic importance. I have also observed the following phenomenon with regard to the position of the head in cerebellar tumors. If the head being held in one position is turned to the opposite side and headache or vertigo or both which usually accompany all intracranial growths will become intense at the time of change of position, the presumption is in favor of the tumor being localized on that opposite side. I have reported six such cases, four of which were verified by autopsies, before the American Neurological Association in May, 1908. (See Special Diagnostic Phenomenon in Cerebellar Diseases, Jour. Amer. Med. Ass., v. LI, 1908, p. 461.) Laruelle (Revue Neurologique, 1906, No: 4) reported a similar observation. He localized the tumor in the left cerebellar hemisphere because the head was inclined to the right. The tumor, however, was found on the right. The record also shows that each attempt to turn the head to the left provoked an unusual pain.

In tumors of cerebello-pontine angle not only the cerebellum and cranial nerves, but also the pons itself is being compressed. The symptoms of the latter will therefore be those of motor and sensory pathways which pass through the pons. Paresis or paralysis, anesthesias or analgesias or else paresthesias, changes of reflexes will be observed. Paralysis is of course observed on the opposite side of the compression. Sensory disturbances as a rule are not frequent, and if present they are very slight. Motor disturbances are also not pronounced in cerebello-pontine tumors, but changes in reflexes are quite frequent. The knee-jerk may be increased on the opposite side and not infrequently diminished on the side of the tumor. The toe phenomenon and ankle-clonus may be present on the side of the plus knee-jerk.

While the three groups of symptoms referable respectively to the cerebellum, cranial nerves and pons are being investigated, the question of the condition of the eyes should be borne in mind. At the beginning of the disease there may be no changes in the fundi so that the diagnosis of a neoplasm may not be entertained especially if the localizing symptoms are very mild. In the case reported below the nystagmus, the paralysis of the external rectus, the engorgement of the veins with edema of the papilla appeared only when the other symptoms became very manifest. At the time the clinical picture consisted only of headache with some vertigo, of a slight staggering, of a slightly exaggerated knee-jerk and of a slight adiadochokinesia of the left hand—at that time there was no trace of changes in the eyes. It is only when the above symptoms became pronounced that the eye began to participate in the pathological process.

The diagnosis of a cerebello-pontine tumor is therefore not to be abandoned in case eye changes are not observed. When unilateral symptoms are repeatedly found no matter how slight they may be and when these symptoms are referable simultaneously to the cerebellum, pons and cranial nerves or at least to the cerebellum and cranial nerves, the presumption is strongly in favor of a neoplasm situated in the angle between the pons and cerebellum. The case reported here is instructive from the diagnostic standpoint, because for a long time the left sided symptoms were so significantly slight that they could easily be overlooked. Their persistence, however, at repeated examinations was of utmost importance for diagnostic guidance. The case is as follows:

J. M., steward, 35 years of age, presents a good family history. He drank heavily. Eight years ago he contracted a chancre for which he was not treated. His three children died at a tender age, from 3 to 5 months.

Several months before he died he began to complain of headache. Four months ago while having these severe headaches on getting up one morning he became very dizzy, but was not unconscious. Headache and dizziness became constant. Soon vomiting set in.

Upon admission to the hospital on March, 1915, he presented the following condition. He complained of a continuous pain in the left supra-oribital area radiating to the temporal region down to the neck on the same side. Pain existed for the last two years but became severe and persistent lately. He also complained of vertigo especially on walking or raising up his head. In walking he had a slight tendency to fall towards the right side; the same tendency was observed when he closed his eyes. The knee-jerks were slightly exaggerated on both sides and only somewhat more marked on the right than on the left side. There was no ankle clonus and no toe phenomenon on either side. Sensibility was preserved in all four extremities and face. Diadochokinesia was very slightly retarded in the left hand when compared with that of the right hand. There was a very slight ataxia in the left hand, but so slight that it could be considered negligible. No other objective symptom was observed. The patient also had some difficulty of micturition and suffered from insomnia. A Wassermann test was positive on serum and negative on spinal fluid.

At that time the diagnosis pointed to cerebro-spinal syphilis. Nevertheless the presence of a few symptoms on the left side of the body, no matter how slight they were, together with the history of headache, vertigo and vomiting—suggested the possibility of a neoplasm in the cerebellum.

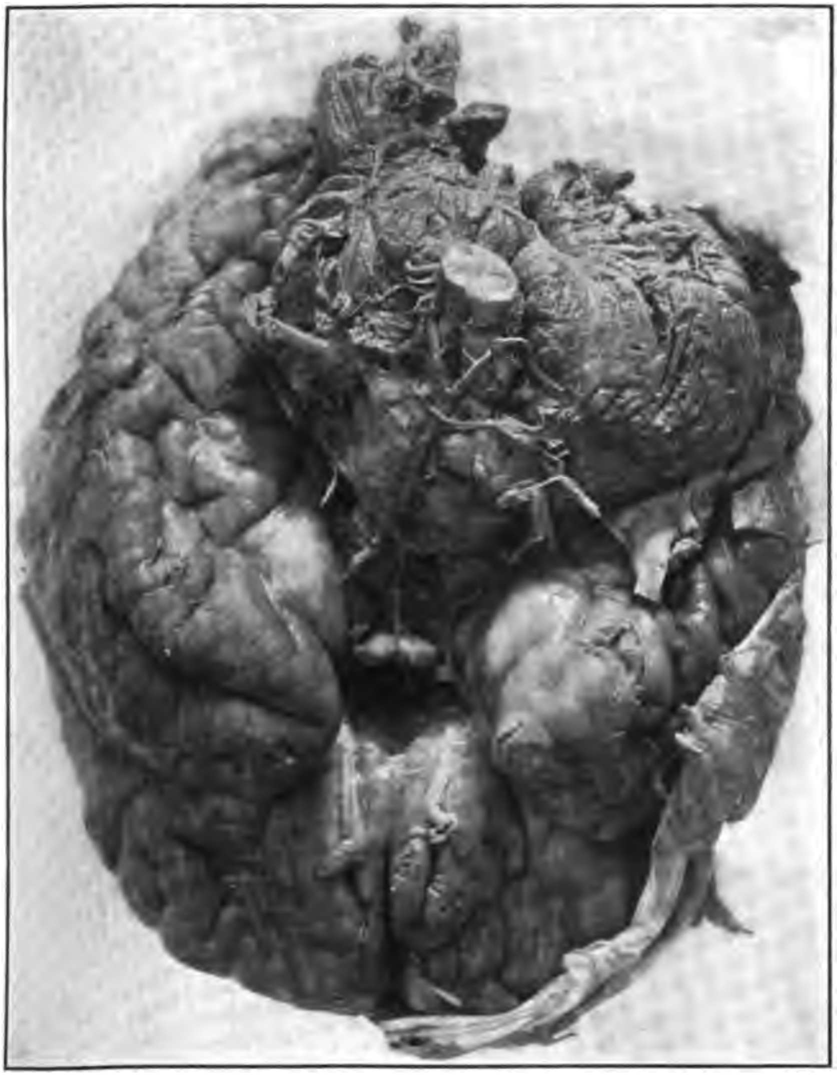
The eyes at the first examination made by Dr. Appleman presented nothing abnormal. The condition remained unaltered for the following two weeks when a slight deviation of the lower part of the face toward the right side was noticed. Very soon a nystagmus appeared in the left eye upon lateral movements of the eyes to the left. Gradually other left-sided symptoms made their appearance. The left side of the tongue was found to be analgesic, the left ear became deaf and the external left ear became analgesic. The external rectus of the left eye became paralytic. Soon the entire left side of the face was paralyzed and it presented a typical paralysis of the peripheral type. The cornea and conjunctiva of the left eye lost the sensibility to pain. In testing the patient for the finger to nose movement dysmetria on the left was very perceptible. The same condition was found in touching his left ear with the left index. Adiadochokinesia is present in the left hand. Flexion and extension of the left forearm is somewhat slow when compared with the right side.

In testing the lower extremities for exact movements dysmetria was noticed in the left foot. The condition of the reflexes is the same as on previous examinations except that in stroking the soles one obtains a more rapid flexion of the toes on the left than on the right.

At that time the eyes began to show changes. There was some slight limitation of eye movement all around. Imperfect closing of left upper lid. In the left eye media was clear, disc oval, edges hazy. Disc color not very good, slightly indented over surface, with moderate swelling. A later examination showed the veins markedly engorged with considerable edema of the papilla in the left eye.

The diagnosis at this time became unhesitatingly in favor of a neoplasm located in the left cerebello-pontine angle. An operation was advised and accepted. Dr. N. Ginzburg performed a subtentorial decompression on the left side. At first the symptoms improved but rapidly the condition grew worse and the patient expired.

At autopsy a hard mass of the size of an English walnut was found in the left cerebello-pontine angle. It was strongly adherent



ROUND CELL SARCOMA

DIAGNOSTIC ELEMENTS OF PONTO-CEREBELLAR TUMORS
Alfred Gordon

to the eighth nerve which was much thickened. The tumor presses posteriorly on the left hemisphere of the cerebellum, the very anterior portion of which is obliterated. The portion of the medulla posteriorly to the pons is deviated to the right. The seventh and sixth nerves on the left side were also deviated as result of pressure by the tumor. Histologically the mass is a round cell sarcoma.

GLIOMA OF THE EYE WITH RAPID RECURRENCE AFTER ENUCLEATION

BY ARMIN NETTLE
New York

During the latter part of July, 1914, the mother brought the patient, a child of three years, to the clinic.

The child was rather anemic and pale and the mother complained of seeing a yellowish spot back of the pupil of the left eye. She had noticed the spot for a few months and as it grew bigger, she sought advice.

I made an examination and found the following condition: The right eye was normal, externally and ophthalmoscopically. The left eye looked normal, except that back of the pupil, which was moderately dilated and reacted to light and accommodation, could be seen a whitish yellowish spot, the typical amaurotic cat's eye. Further dilatation of the pupil was made and inspection was made of the tumor.

With the aid of the electric ophthalmoscope, vessels were seen running across its surface. The eye was also transilluminated and a darkened shadow could be seen. No light reflex being seen through the pupil. There was apparently no vision in this eye.

The history of the case was then inquired into. The child was born in normal labor, had been weaned at the ninth month. Had suffered no illnesses. No cough which would lead to any suspicion of pneumonia and metastatic choroiditis.

The family history was also negative, so from examination and the history of the case, a positive diagnosis of glioma was made and immediate enucleation was advised and insisted upon. The mother consented and the operation of enucleation was performed the following day.

I enucleated the eyeball and cut the optic nerve as far back as possible. The orbital wound healed kindly and the child was discharged from the hospital one week later.

Three weeks later the child returned to the hospital, and I noticed at the inner canthus of the eye, a swelling the size of a pea which I diagnosed as a recurrence of the glioma. I advised X-ray treatment and sent the child to a radiologist. It did not show any improvement, just the contrary, it rapidly increased in size until it finally filled the oribital cavity and protruded from one to two inches. It bled readily on touch. Another operation on the growth I deemed inadvisable after consultation.

Two and a half months after enucleation the child began to show cerebral symptoms, vomiting, convulsions, twitchings of various muscles, a condition of apathy which ended in a comatose condition and about three months later it died in coma.

Pathological report:—The section is cut below the pupillary opening and, therefore, does not permit of a study of the anterior portion of the eye, nor of the optic nerve.

The globe is nearly filled with the tumor growth and springs from the retina, which is detached and disorganized. The choroid shows secondary invasion by the tumor cells, and at one spot the tumor is underneath the choroid between it and the sclera. This is significant.

Careful study of the section reveals a cluster of the tumor cells outside of the globe. In the posterior portion of the eye near the optic nerve in the midst of the posterior ciliary nerves and vessels, there is an area which shows the cells arranging themselves about these tissues.

The tumor is composed of cells and blood vessels and no intercellular tissues are seen. The cells are mostly round and show rapid division, and a marked necrosis of the cells is noted near the periphery of the tumor. There are so many new formed blood paths around which are seen the largest sized cells. These cells have a single nucleus and are partly granular and show rosettes (Wintersteiner) very nicely. The best examples of the glioma cells are seen in these rosette formations and the further from the rosettes the cells get, the more broken up they become and the tumor becomes granular in appearance. This is due to a necrosis of the cells and the setting free of the glioma cell granules.

There is a great amount of hemorrhage in the tumor. It is impossible to say which layer of the retina the tumor springs from, but it is usually the interval granular layer that gives birth to the gliomas. The presence of the glioma cells outside of the globe makes it highly probable that this tumor will return.

The gross specimen of the eye on section, shows the tumor occupying the entire posterior chamber of the eye up to the posterior surface of the lens. It is yellowish gray in appearance and shows granular areas, especially in the center. It seems to spring from the optic disc.

This case illustrates the necessity for the early diagnosis of glioma and this, in most cases, is impracticable. It is only after the tumor has grown to sufficient size, that the mother noticed the yellow gray reflex back of the pupil, is there any idea of there being anything wrong with the eye and this is well brought out in the pathological report.

The presence of glioma cells was demonstrated outside of the globe which made it highly probable as to a recurrence of the growth in the extra-orbital tissues and the very rapid return. The very early age of the patient makes it impossible of diagnosis.

The first clinical characteristic of the tumor is that it is met with exclusively in early life. Occasionally it has been observed as late as the twelfth year, but ordinarily it occurs during the first four years. According to Wintersteiner, the majority of children have not completed their third year.

The second clinical characteristic in these cases, *i.e.*, the recurrence in various members of families, was not noticed in this case.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

Source of Error With Esbach's Albuminometer—H. E. JONES, Glasgow Med. Jour., Jan., 1916.

In testing a urine with Esbach's reagent it is important to ascertain how much alkalinity is present, for if this be too high, the amount of citric acid in the reagent will not be sufficient to acidulate the urine to the extent required, and therefore the excessive alkalinity will be a source of error in the test. SACHS.

Carmin Test in Infants and Children—A. HYMANSON, Am. Jour. Dis. Child., Feb., 1916.

Author states that a complete passage of carmin, when given by mouth, of less than fourteen hours is abnormal. In children with accidental catarrh, the passage time varied from 9 to 12 hours, and in hyperemic and ulcerated forms of catarrh from 3 to 8 hours. The main point deduced is that (since the more severe the condition, the higher up the lesion) these quick passages have a certain diagnostic value. If carmin given by mouth does not appear in the stools within a definite interval, there might be good reason to suspect the existence of pyloric obstruction. Carmin is givn in children in doses of ½ to 2 grains. SACHS.

Acidosis in Children—E. C. WILLIAMS, Brit. Jour. Child. Dis., Nov., 1915.

Acetone and diacetic acid were present in 3 cases with symptoms suggestive of meningitis in which the cerebrospinal fluid showed no cytological changes. Two cases of mussel poisoning and one case of ice-cream poisoning gave a marked reaction for acetone. In one case acidosis preceded an attack of pneumonia, in another it occurred during the course of the pneumonia, and in a third case pneumonia developed in a patient in which acetone followed the disappearance of sugar from the urine. In one case acidosis was present in which the post-mortem revealed suprarenal hemorrhage. A case which had symptoms suggestive of intussusception gave a marked reaction for acetone and the symptoms cleared up under treatment with alkalies. SACHS.

Russo Reaction—M. KAHN and L. WECHSLER, Med. Rec., Jan. 15, 1916.

Russo's reagent consists of a 0.1 per cent. aqueous solution of methylene blue. Several drops of this reagent are added to a few c. c. of urine. A positive reaction is characterized by the fact that the urine assumes a mint-green or emerald-green color. If any trace of a bluish tint is present, the reaction is negative. In most cases of typhoid, this reaction appears toward the end of the first week. The incidence of this test in typical cases of typhoid fever is in about 60 per cent. of the cases, equalling the incidence of the positive diazo reaction in this disease. A positive Russo reaction was obtained in ten per cent. of cases of tuberculosis. SACHS.

Acid-Fast Bacilli and Joint Disease—H. KELLER and A. J. MORAVEK, Med. Rec., Nov. 20, 1915.

Keller and Moravek have come to the conclusion that the acid-fast bacilli in the feces of patients suffering with joint disease which resist decolorization with 25 per cent. nitric acid water solution followed by alcohol (80 per cent.), are tubercle bacilli. This acid-fast bacillus is viable, able to grow on culture media and when such cultures are injected into guinea-pigs, they produce tuberculosis. Under normal conditions there are no acid-fast bacilli in the feces. They are present only in active cases of joint disease and not in cured cases. SACHS.

Tonsilar Endamebiasis and Thyroid Disturbances—J. S. EVANS, W. S. MIDDLETON and J. A. SMITH, Am. Jour. Med. Sc., Feb., 1916.

Out of 38 cases of typically diseased tonsils in goitrous individuals, 97 per cent. were found to harbor endameba gingivalis. Sixteen of these cases were given emetin hydrochloride and after such treatment, 13, re-examined, showed an absence of endamoebae.

SACHS.

INFECTIOUS DISEASES

Method of Detecting Spirochetes—H. C. COLES, Brit. Med. Jour., Nov. 27, 1915.

Coles stains a film containing the spirochetes with the Giemsa or Leishman stain and examines it dry, not mounted in any medium, with dark ground illumination, using a dry lens of medium power. He states that by such a method the spirochetes are as easily detected as tubercle bacilli in the sputum. Structures colored with a fluorescent dye, when examined with a dark ground, stand out prominently, and if the medium in which they are be of low refractive index, they may be easily seen with comparatively low power. If mounted in balsam, cedar oil, parolein, or water, they are only seen with the greatest difficulty. SACHS.

Complement-Fixation Reactions of Bordet-Gengou Bacillus—M. P. OLMSTEAD and O. R. POVITZKY, Jour. Med. Research, Jan., 1916.

By means of complement-fixation tests authors have been able to confirm the separation by morphological and cultural characteristics of the typical Bordet-Gengou bacillus from atypical strains of the same and from the influenza bacillus. Twelve strains of the B. pertussis isolated by them from patients with pertussis have given complement-fixation reactions similar to each other and similar to the true strains obtained from other laboratories. SACHS.

Neutrophile Leukocytes in Malaria and Yellow Fever—J. W. S. MACFIE, Annals Trop. Med. and Parasitology, Dec., 1915.

A slight shift to the left of the Arneth count is observed in the blood of healthy Europeans in West Africa. In malarial fever there is a marked shift to the left. In yellow fever there is a great shift to the right of the Arneth count. SACHS.

Meningococci in Nasopharynx—J. MCINTOSH and W. E. BULLOCK, Lancet, Nov. 27, 1915.

Authors use serum agar (nutrient 3 per cent. agar, 3 parts; horse serum, one part) as a medium for the development of the meningococcus. They are of the opinion that the meningococcus carriers are less frequent than is generally believed. SACHS.

Röntgen-Ray in Lobar Pneumonia in Childhood—H. H. MASON, Am. Jour. Dis. Child., March, 1916.

Thirty-seven cases of lobar pneumonia were studied by the Röntgen rays. All 37 cases showed a definite shadow. The shadow was always so placed that it touched the pleura at some point. The early shadows were triangular in shape, with their bases on the pleura and their apices separated from the region of the root by normal lung. In their later development the shadows extended inside and became uniform from the periphery to the root of the lung. When the shadow involved this entire stretch, bronchial voice and breathing were present, but not otherwise. Author believes that the inference is justified that the dimensions of the shadows correspond to the extension of consolidation. SACHS.

Röntgen-Ray Examination in Tuberculosis of Infancy—C. H. DUNN, Am. Jour. Dis. Child., Feb., 1916.

The Röntgen-ray examination was positive in every one of 13 cases of tuberculosis in infancy. There were several cases in which it gave the only positive finding. Tuberculous bronchopneumonia was correctly diagnosed from the Röntgen-ray in several cases, but not in all those in which it was found post-mortem. SACHS.

Röntgen-Ray Localization of Dysenteric Ulceration—C. J. GLASSON, Proc. Royal Soc. Med., Dec., 1915.

In acute cases of dysenteric ulceration, radiography will not be of much assistance, but in chronic cases it will be of great assistance both to the physician and surgeon, as it will give a clear picture of areas where the ulcerated patches are situated. Before a radiogram is taken, the patient should be given bismuth subnitrate in doses of 60 grains every two hours during the day for at least six days. On the sixth day a radiogram should be taken, then the bismuth should be discontinued and liquid paraffin given in two-dram doses every two hours for twenty-four hours. Then the second radiogram should be taken and this second one will show where any ulcerative patches may be situated. SACHS.

Bacteria of Gangrenous Wounds—H. R. DEAN and T. B. MOUAT, Brit. Med. Jour.,

Streptococci were isolated in nearly every one of the 18 cases investigated by authors. Staphylococci were present less frequently, and then usually in small numbers. B. coli were present in only 4 cases, and B. proteus in 1 case. Three varieties of anaerobic bacteria were isolated from these cases. The bacillus of malignant edema was present in 15 cases, B. aerogenes capsulatus in 13 cases, and B. tetani in 4 cases. SACHS.

Bacteriology of Septic Wounds—A. FLEMING, Lancet, Sept. 18, 1915.

Author found three groups of bacteria in septic wounds: (1) spore bearing microbes of fecal origin: (2) non-sporing microbes of fecal origin; (3) pyogenic cocci. Included in the first group are B. tetani, B. aerogenes capsulatus of Welch, and certain putrefactive organisms referred to as bacillus X. and bacillus Y. These are all found in the intestines of man and animals, or in the soil which has been manured with animal excrement. The second group includes the non-sporing bacteria of fecal origin: streptococci, B. proteus, B. coli group, and "Wisp" bacilli. The streptococci are present in almost every wound up to a late stage. Fleming assumes that the streptococci are of fecal origin, because of their association with other organisms of known fecal origin and their appearance early in the wound. The third group comprises especially the staphylococcus and the streptococcus. The staphylococcus is almost the only organism frequently occuring in wounds which is not common in animal feces. The B. X. is a straight bacillus, 5 microns long and 1 to 1½ microns broad, non-motile, Gram-positive, and sporing freely in all mediums. The B. Y. is a long, slender Gram-negative bacillus, freely motile, which shows close to or at one extremity an elongated spore. SACHS.

Tonsils and Chronic Cervical Adenitis—H. GARDINER, Lancet, Oct. 2, 1915.

Author states that the tonsils are infected in 80 per cent. of cases of chronic cervical adenitis in which no obvious source of infection is present. Small fibrotic tonsils are more dangerous than large tonsils. Tubercle bacilli are more frequently present than in simple cases of enlarged tonsils. A large proportion of the so-called chronic tuberculous glands are in reality chronic septic glands by reason of the presence of other organisms than the tubercle bacilli in these cases. SACHS.

Epidemic Abortion Reaction in Children—J. P. SEDGWICK and W. P LARSON, Am. Jour. Dis. Child., Sept., 1915.

Contagious abortion of cattle has been known to stockmen and dairymen the world over for the past century or more. The disease is due to a short Gram-negative, non-motile bacillus, called bacillus abortus. A variation of complement in children with the use of the bacillus abortus as antigen may be found in the blood serum of those consuming milk from cattle with this infection. Newborn children who have been fed at the breast only, do not show the reaction. One baby three weeks old, after having been off the breast two weeks and on a food containing cow's milk, gave a positive reaction. The finding of a positive reaction in children with large spleens is suggestive. The blood of children giving positive Wassermann reactions may show no deviation of complement when the bacillus abortus is used as antigen. Patients with many of the diseases of childhood, including rickets, give negative reactions. For the practising physician, the importance of breast feeding and the desirability of the heating of all cow's milk administered is emphasized. SACHS.

Sclerema Neonatorum—W. S. PATERSON, Quart. Jour. Med. (London), July, 1915.

Sclerema neonatorum is a definite disease. It may vary considerably as to degree in the extent and severity of its manifestations, and also as to the gravity of its prognosis. It is found shortly after birth. The most characteristic feature is the palpable subcutaneous induration, with which there may be associated abnormalities of temperature, most notably in the direction of its being subnormal. Visceral changes may occur as complications, in the form of inflammations, hemorrhages, or serous oxudates. The etiological factor is probably some microbic infection. SACHS.

RESPIRATORY AND CIRCULATORY ORGANS

Parapharyngeal Abscess—H. HEIMAN, Am. Jour. Dis. Child., Aug., 1915.

There occurs, especially in children, a form of abscess which is occasionally mistaken for a retropharyngeal abscess, but which has a separate and distinct entity. It may be called a parapharyngeal abscess as its site is in the lateral columns, that is at the side of the pharnyx. The origin of this abscess is from the superior chain of the deep cervical glands, which are situated along the course of the carotid artery, as distinguished from the retropharyngeal glands, situated in the circumscribed retropharyngeal space, just lateral to the mid-line, and in front of the prevertebral muscles. These abscesses are, as a rule, cured only by external operation. This form of abscess produces bulging near the lateral pharyngeal wall, if at all internally, but rarely in the middle line; it nearly always displaces the tonsil towards the middle line, and a swelling may appear externally just below the angle of the jaw. It is rarely accompanied by any marked local changes in the appearance of the pharynx. SACHS.

Bronchial Glands—W. W. HOWELL, Am. Jour. Dis. Child., Aug., 1915.

Author accepts as a positive D'Espine sign, a change in character of the whispered voice or expiration at or below the third dorsal spine. Dulness to the right of the vertebral column at the level of the spine of the scapula is definite and significant of enlarged glands and seems to agree pretty well with the intensity of the D'Espine sign. In no case with the above two signs did the röntgenogram fail to show a shadow. For purposes of diagnosis it is well to think of the bronchial glands as of those up and down the trachea, and those at the root of the lungs. The upper set become enlarged from draining the upper regions and give a narrow chain which is sufficient to transmit the bronchial character of the sounds, but does not give dulness. A positive D'Espine with interscapular dulness means thickening at the root of the lungs, secondary to a process in the lungs which in infancy means tuberculosis, and, in older children, if accompanied by malnutrition and anemia, is probably tuberculous. SACHS.

Diagnostic Significance of D'Espine Sign—H. F. STOLL, Am. Jour. Med. Sci., Sept., 1915.

Whispered bronchophony in the interscapular space (D'Espine's sign) is indicative of a pathological process at the hilum of the lung. This may be due to enlarged glands, the result of malignancy, leukemia, Hodgkin's disease, syphilis, or any infectious disease of the lungs. Author has also seen it several times in aortic aneurism.

Its presence in the delicate child is exceedingly suggestive of tuberculous involvement of the tracheobronchial glands. Occasionally enlargement of the bronchial glands is present when there is no change in the whispered voice. In old people, in whom the usual physical signs of pulmonary tuberculosis are sometimes exceedingly difficult to elicit, the character of the whispered voice in the interscapular space should always be ascertained, as a well marked D'Espine sign speaks for tuberculosis rather than for chronic bronchitis, or emphysema. The diagnosis of clinical tuberculosis, however, rests on the sum total of our physical signs and symptoms and not on one isolated sign. SACHS.

Pulmonary Affections and Gout—A. MAYER, Zeitschr. f. klin. Medizin, Vol. LXXXI, Nos. 5 and 6.

Among 40 patients with chronic bronchitis and bronchial asthma, who never had an attack of gout and never had presented any of the general symptoms of gout, the purin metabolism was characteristically disturbed in 4 patients. In these the glycocol excretion curve was typical and the uric acid retention proved by means of the Umber-Retzlaff test. In 5 more patients all these earmarks of an arthritic diathesis were present, but neither tophi or creaking in the joints could be detected. In all the cases there existed a chronic bronchitis with an enduring mild asthmatic condition, a mild emphysema and a mild chronic expiratory dyspnea. In some of the cases exacerbations of the respiratory condition were due to the same causes as the typical attack of gout, as small doses of alcohol, ingestion of liver, etc. Author succeeded repeatedly in experimentally producing attacks of pulmonary gout by the administration of sodium nucleinate, and the typical behavior of uric acid in the blood and urine and the glycocol excretion. Nearly all the relatives of the patients had genuine gout. Exudative diathesis was frequently present in the children of the patients. The reason why uratic deposits were not found in the lungs is that pulmonary tissue has very little affinity for uric acid and that even when it is injured, it has very little affinity for the same. The treatment of general gout is well adapted for gout of the lungs.

WESTERN.

Blood Pressure Determinations—R. I. LEE, Bost. Med. and Surg. Jour., Oct. 7, 1915.

Author examined 662 normal young men averaging 143 pounds in weight. The average systolic pressure was 120 mm. Hg., and the average diastolic pressure was 80 mm. Hg. Eighty-five of these cases presented a systolic blood pressure of over 140 mm. Hg. The highest reading was 180. The diastolic pressures were much more uniform. Only 5 cases had a pressure over 100. Of the 662 men, 5.1 per cent. had albuminuria. Seventy-three patients had a lymphocytosis of over 50 per cent. SACHS.

Adolescent and Beginning Atherosclerosis—S. SALTYKOW, Korrespondenzblatt f. Schweizer Aerzte, Nos. 34 and 35, 1915.

The so-called fatty state of the intima in the arteries of children and adolescents, especially in the aorta, is nothing else but the beginning of atherosclerosis. All recent investigations point with certainty to the conviction that atherosclerosis starts in very early life, that it is latent for a long time, and that it ensues together with the differentiation processes of the intima. MILL.

Hemoptysis Due to High Tension—F. DE H. HALL, Lancet, Aug. 14, 1915.

Author cites 5 cases of hemoptysis which he is firmly convinced were due to high blood pressure. SACHS.

The Diagnosis of Cardiac Diseases—H. ROSIN, Deutsche med. Wochenschr., Sept. 2, 1915.

The older clinico-physical methods employed in the examination of the heart continue to be as valuable as ever. Blood pressure determination, palpatory percussion, röntgenoscopy and röntgenography and the Wassermann reaction supplement the physical methods. The electrocardiogram, it is true, is an important diagnostic adjuvant, which, however, is as yet not of sufficient practical value.
MILL.

The Mechanism of Paroxysmal Tachycardia—S. LA FRANCA, Zeitschr. f. klin. Medizin, Vol. LXXXI, Nos. 5 and 6.

Paroxysmal tachycardia differs from other types of tachycardia by reason of peculiar, entirely distinct symptoms. It develops with the mechanism of the early contraction. According to the point of origin of the abnormal irritation there exists an auricular, ventricular or atrioventricular form or a mixed form may be produced. The appearing intermissions belong to the mechanism of the early contraction. They may be of different origin, however. The intermittances may originate in the heart itself or in the irritative condition of the accelerans. In no case, however, can the influence of the extracardial nerve apparatus be entirely excluded.
WESTERN.

ALIMENTARY TRACT

Acute Non-epidemic Inflammation of the Sublingual Glands in Children—H. NEUHOF, Am. Jour. Dis. Child., Aug., 1915.

A group of cases of sublingual gland inflammation in otherwise healthy children is presented, differing in clinical picture and in etiology from the hitherto described acute non-epidemic lesions of these glands. The usual causes of inflammation of the sublingual glands are not in evidence. The characteristic features are: Sudden onset of sublingual and submental swelling with mild febrile manifestations; scattered red spots in the mucous membrane over the swelling, interpreted as inflammation of the orifices of the sublingual ducts; rapid increase in size, often to grotesque proportions, of the moderately painful submental swelling, frequently accompanied by pronounced reddening of the overlying skin; early recession of the sublingual swelling, contrasting with the persistence of the submental tumor for one to three weeks; fever slight and general condition good throughout the course; no complications, sequelæ or recurrences. SACHS.

Acidity of the Nursling's Stomach—R. HESS, Zeitschr. f. Kinderheilkunde, Vol. XII, No. 6, 1915.

The acidity in the gastric contents of the nursling ascends with the development of the organism. In the ninth month the degree of acidity, which is essential for the activity of pepsin, is attained. MILL.

Detection of Pepsin and Trypsin in the Fasting Stomach—G. DORNER, Deutsches Archiv f. klin. Medizin, Vol. CXVII, No. 6.

Pepsin can most always be demonstrated in the contents of the fasting stomach by means of the Edestin method, and trypsin by the casein test. The secretion of the pancreas probably ensues in the same manner as that of bile and saliva, viz., in small amounts, but incessantly. Pepsin is almost always contained in the fasting stomach. However, it is occasionally destroyed on account of a higher degree of alkalescence. While pepsin may not be found in the test meal it may frequently be demonstrated in the urine. WESTERN.

Opaque Meal Shadows in Diseases of the Stomach—J. H. CODD, Practitioner (London), Aug., 1915.

Excessive motility may be defined as the picture seen when a wave passes along a shadow and cuts into it for a distance of half of its depth or more; when a wave passes along from one end to the other in ten seconds or less; and when the long axis of the shadow moves through a considerable angular distance or executes vermicular movements. If the stomach shows excessive motility immediately on the reception of the meal, or in four hours, and the stomach is clear in six or eight hours, there is probably peptic ulcer. If there is no excessive motility immediately, but it shows itself in four hours, and the stomach is not empty in eight hours, there is probably pyloric obstruction, especially if there is excessive motility in eight hours, as well as in twenty-four hours in an unemptied stomach. The hour-glass shape is fairly typical and should show a definite neck or isthmus. If there is mere contraction with both sacs in contact, it is probably spasmodic and should be watched for signs of change. If Barclay's duodenal sign is present, there is probably ulcer of the duodenum, either a surgical or a superficial erosion, impalpable from the outside. SACHS.

Pyloric Hypertrophy in Newborn Infants—F. L. WACHENHEIM, Am. Jour. Dis. Child., Aug., 1915.

Pyloric spasm is a probable causative factor in the development of true organic obstruction. A retrogressive change normally takes place between the third week and the third month, in the course of which the very perceptible normal thickening of the pylorus of the newborn infant gives way to a condition in which the borders of that organ are macroscopically undistinguishable. The thickness of the pyloric wall in the normal infant of two months is 2 to 2.5 mm.; in the newborn it is greater, 3 to 4 mm.; in established hypertrophic stenosis it is fully 5 mm. The average newborn infant bears within itself the latent tendency to develop pyloric hypertrophy. Pyloric thickening was noted at autopsy in 4 consecutive newborn infants, and while it would be presumptuous to state from such a small series that pyloric thickening is universal, it seems to show that at least it is present in the majority of cases.

SACHS.

Gastric and Duodenal Ulcers—C. GRAHAM, Bost. Med. and Surg. Jour., Oct. 7, 1915.

Seventy-two per cent. of duodenal cases seen at the Mayo clinic in the past 9 years had fairly regular symptoms and 71 per cent. of gastric cases were so clearly gastric, that it was not difficult to make a diagnosis. Pain is the one constant diagnostic factor in all peptic ulcers. In 23 per cent. of duodenal ulcers the pain comes one-half to two hours after the taking of food, and in 77 per cent. of the cases in 2 to 5 hours. In gastric ulcers the pain comes on in one-half to two hours in 50 per cent. of the cases and in 2 to 5 hours in the remaining cases. Eight per cent. of the patients with duodenal ulcers and 19 per cent. with gastric ulcers had pain within one-half to one hour after eating. Ease from posture (lying down) is more frequent in gastric ulcer than in duodenal ulcer. Tenderness to touch was observed in 41 per cent. of the duodenal and in 40 per cent. of the gastric ulcers. A history of hemorrhage by mouth was recorded in 18.5 per cent. of duodenal and in 25 per cent. of gastric cases. Blood from the bowels was similarly recorded in 18 per cent. of duodenal and in 24 per cent. of gastric cases. Perforation was observed about equally in both classes of ulcers, and pain in the back was also about equal. SACHS.

Pancreatic Secretion—M. Einhorn, Med. Rec., June 12, 1915.

In order to ascertain the condition of the duodenal contents in health, several apparently perfectly healthy persons were examined with regard to the state of their pancreatic secretion. They took the duodenal tube either at night before retiring, or between 4 and 5 A. M., with a glassful of water. They then slept, rose at 8 A. M., and called at my office at 9 A. M. in the fasting condition, when duodenal contents were obtained. I am greatly indebted to them for their willingness to be thus examined. The amount of ferments in normal individuals fluctuated as follows: Amylopsin, 4 to 8 mm.; steapsin, 2 to 5 mm.; trypsin, 0.5 to 5 mm. The average figures were: Amylopsin, 6 mm.; steapsin, 3.5 mm.; trypsin, 2.8 mm. The alkalinity, as determined by one-tenth standard solution of hydrochloric acid with methylorange as an indicator, varied between 15 to 40. The rennet ferment was present in all. During the last three years author has examined 170 patients with regard to their duodenal contents, making about 275 analyses of the pancreatic secretion. The examination took place either in the fasting condition of the patient or after tea and sugar or clear bouillon, ordinarily about one to one and a half hours after the ingestion of the latter into the stomach. The duodenal tube was introduced either the night previous or two or three hours preceding the examination. The contents were obtained first by aspiration, then being followed by syphonage. The quantitative examination of the amounts of the different ferments was determined by agar tubes. The alkalinity was determined in 57 patients and averaged 22. Ordinarily it fluctuated between 10 and 30. One patient showed an alkaninity of 80 which was exceptional. As far as can be seen there is no relation between gastric acidity and pancreatic alkalinity. In looking over the results obtained by these examinations there exists a noticeable independence between the three different ferments with regard to quantity in the same individual—one ferment may be present in large amounts, while the other two may be present in small amounts or may be absent. The quantity of one ferment does not permit us to presume the amount of the two other ferments. It is thus necessary to test for each of the three ferments separately. This is accomplished in the most convenient way by means of agar tubes. The pancreatic secretion is subject—similar to the gastric juice—to functional anomalies or deviations from the normal. The juice may contain an overabundance of ferments or too small an amount of them. Again there may be hypersecretion or greatly diminished secretion. While in the gastric juice the functional activity is generally reckoned by the amount of hydrochloric acid present, there has as yet no definite substance of the pancreatic secretion been selected for this purpose. The trypsin ferment being the most important ingredient of the pancreatic juice, author suggests to use it as a gauge for the functional activity of this gland. The following terms may then be advantageously used: Eupancreatism=normal function; all three ferments present, trypsin showing the normal quantity (1 to 4 mm.). Hyperpancreatism=increased activity; all three ferments present, trypsin existing in excess (above 4.0 mm.). Hypopancreatism=diminished activity; the three ferments present, trypsin decreased (below 1 mm.). Dyspancreatism=disturbed function; one or two of the three ferments are absent. Heteropancreatism=varied function; the presence and amount of ferments showing no constancy, but variations every now and then. SACHS.

Intermittent Ileus and Movable Kidney—HÜGELMANN, Münchener med. Wochenschr., Aug. 17, 1915.

Description of a case in which a movable kidney gave rise to an intermittent compression ileus. The direct cause of the ileus was the appearance in regular intervals of a hydronephrosis. MILL.

Calcium Content of Bile and the Formation of Biliary Calculi—L. LICHTWITZ and F. BOCK, Deutsche Med. Wochenschr., Oct. 14, 1915.

The calcium content of human bile in the gall-bladder fluctuates between 85 and 352 milligrams in 1000 cc. bile. In cholecystitis there is, as a rule, no increase of the calcium content. The calcium content in the bile of a fresh biliary fistula varies between 65.1 and 83.9 milligrams in 1000 cc. MILL.

Appendicitis as Sequela of Tonsillitis—H. B. ANDERSON, Am. Jour. Med. Sci., Oct., 1915.

Since the appendicular involvement in cases of tonsillitis may be only a part of a generalized infection, it is evident that the gravity of such cases is out of proportion to the local symptoms. These cases have a tendency to be atypical in their clinical course. After smouldering, they may suddenly develop fulminating symptoms. SACHS.

Extraperitoneal Appendix Vermiformis—A. STRAUSS, Surg. Gynecol. and Obstet., Sept., 1915.

Author analyzed 5 cases of extraperitoneal appendices. Such cases are not uncommon. One should be on the alert and look for them and not think that the appendix has sloughed away. Disease of an extraperitoneal appendix may give the typical appendicitis symptomatology or it may simulate perinephritic or psoas abscess. It may produce a subphrenic abscess or a fecal fistula. Fetid pus from an abscess in the right lumbar region should suggest disease of an extraperitoneal appendix. SACHS.

Peritoneal Exudates—R. B. CARLSON, Brit. Jour. Surg., July, 1915.

Examination of peritoneal exudates is of great prognostic value in cases of spreading peritonitis, as it indicates the amount of bacterial invasion and the extent to which the reactive forces of the peritoneal cavity have been, or are being developed. In considering the significance of the relative number of the various cells, their phagocytic activity, and their healthy or degenerated character, the duration of the bacterial invasion must always be borne in mind.

SACHS.

Bacteriology of Peritoneal Exudate—L. S. DUGEON and B. C. MAYBURY, Lancet, Aug. 21, 1915.

In the examination of 23 peritoneal exudates bacteria were absent in 13 and present in 10. In the 10 positive cases cocci (diplococci or streptococci) were present in each instance, and also bacilli in 2 fatal cases. Streptococci occurred more frequently than any other bacteria. SACHS.

NERVOUS SYSTEM

Migraine and Ocular Defects—G. F. C. WALLIS, Practitioner (London), Aug., 1915.

Author cites 4 cases of ordinary and atypical migraine attacks in which complete relief was obtained by properly fitting glasses. In these cases slight compound hypermetropic astigmatism, slight astigmatism and presbyopia, hypermetropic astigmatism, myopic astigmatism, and hyperphoria were the causative factors in the production of the migrainous attacks. SACHS.

Injuries of Superior Longitudinal Sinus—G. HOLMES and P. SARGENT, Brit. Med. Jour., Oct. 2, 1915.

Authors have observed 70 cases of injury to the longitudinal sinus, or of disturbance of the circulation of its venous tributaries in its immediate neighborhood. In some of these cases the brain was not injured, but in others there were gross cerebral lesions. When the disturbance of the sinus is severe enough to cause paralysis, the distributation of the palsy and its relative severity in the different segments of the limbs is peculiar and differs from the ordinary cerebral palsies. The proximal segments of the upper limbs are most seriously paralyzed, and the weakness diminishes distalward. In the ordinary hemiplegia due to vascular legions, the distal segments of the upper limbs are almost invariably more affected than the proximal ones. Speech is never affected in pure sinus injuries. The distribution of the palsy in the legs is the converse of that of the arms. It is always the distal segments that suffer most severely. Rigidity is almost always associated with the palsy. It may come on within 24 hours after the injury.

SACHS.

Clinical Distinctions between Cerebral Intoxication, Meningism and Meningitis—H. HEIMAN, Arch. Pediat., Aug., 1915.

In distinguishing clinically between cerebral intoxication and meningitis, the following points must be borne in mind: (1) In intoxication there is always an etiologic factor, to which the meningeal signs are secondary. Occasionally the cause is difficult to find, but thorough investigation and observation will often reveal one of the infectious diseases or other causal factors. (2) The signs in cerebral intoxication are significant of a general cerebral irritation; in primary meningitis there are, in addition, distinct focal signs. In intoxication we may have headache, photophobia delirium, muscular twitchings, possibly general convulsions; in meningitis we may also have ptosis of the eyelids, unequal pupils, strabismus, facial palsy, even hemiplegia and other focal manifestations. (3) Lumbar puncture is, of course, the most important and definite means of differentiating the two conditions. In intoxication the fluid is sterile and clear; in meningitis bacteria will, in most cases, be found in the fluid either by smear or culture, and the appearance of the fluid is changed from the normal. The cytologic examination of the fluid is also significant, as in the intoxication cases no variation from the normal occurs, whereas in meningitis various changes are present, depending upon the type of the infection, i. e., whether meningococcal, pneumococcal, influenzal, tuberculous, etc. When the ordinary clinical and laboratory methods are not sufficient to distinguish between the two above-mentioned conditions, lumbar puncture is not only justifiable but necessary, even in the presence of an acute infection like pneumonia or typhoid fever. The procedure itself is harmless, except in very unusual cases, and is of the greatest diagnostic value.

SACHS.

Alexia—S. P. GOODHART and H. CLIMENKO, N. Y. Med. Jour., June 19, 1915.

A case report which should be read in the original. Alexia, pure and simple, is an inability to read, and may be a part of the disturbed function of ability to recognize pictures or drawings. The visual memory center being probably in that part of the parietal lobe known as the angular gyrus, a lesion of this area in the left hemisphere, in right handed individuals, would be likely to cause alexia. A lesion of the association fibers between the cuneate lobule of the occipital lobe and the angular gyrus, would likewise cause alexia; associated with this, we should expect to find changes in the retinal field because of the proximity of the fibers of the optic radiation, as these fibers pass from the chiasm back to the occipital lobe.

Because of the limited disturbance the most likely lesion, as probably exists in our case, is of a vascular nature; this frequently being part of a general arteriosclerotic process, other cerebral symptoms are likely to complicate the picture. Pure alexias are not frequently observed, and in a fair number reported in the literature there are also present other forms of aphasic symptoms. The case observed by authors represents perhaps as pure a form of alexia as one meets clinically. SACHS.

Menstruation and Psychosis—P. JOLLY, Archiv f. Psychiatrie u. Nervenkrankheiten, Vol. LV, No. 3.

A menstruation psychosis as a clinical entity does not exist. However, there are cases which show certain connections with menstruation. In most of these cases one has to deal with psychic disturbances ensuing before menstruation. Later on the connection with the menstruation disappears. In some instances the psychosis supervened prior to the first menstruation, seemingly appeared every four weeks and disappeared after the girl had started to menstruate regularly. The menstruation psychoses may be in the form of mania, in that of hebephrenic and katatonic attacks, in that of amentia and hysteria, and, more rarely, in that of melancholic disturbances. Dipsomania may also have some connection with menstruation. The frequency of menstruation psychoses is frequently exaggerated. The influence of menstruation upon chronic psychoses is not very marked. Menstrual disturbances in psychoses are frequent. Amenorrhea is very often encountered in acute psychoses.

WESTERN.

Death as the Consequence of the Epileptic Attack—O. HEBOLD, Archiv f. Psychiatrie u. Nervenkrankheiten, Vol. LV, No. 3.

Sudden death in epilepsy is caused (a) by the attack per se (cardiac weakness, cardiac rupture, cerebral hemorrhage); (b) by a fall giving rise to lethal injury (breaking of the neck or a vertebra, fracture of the skull); (c) by suffocation on account of the posture (lying on abdomen or face); (d) the patient dies by aspirating certain material (remnants of food during meals, water when bathing) or he burns by falling into a fire; (e) at the end of an attack, when in a condition of disturbed consciousness, he may commit suicide. Death after an attack may ensue (f) on account of the attack itself by cerebral hemorrhage; (g) by a fall upon the head during an attack (hemorrhage of the brain and meninges); (h) by a fall into a fire and hot liquids (burning, scalding); (i) by infection of the wounds sustained during an attack.

WESTERN.

URINARY ORGANS—MALE GENITALIA

Opsiuria—L. v. SZÖLLÖSY, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVII, No. 2.

Opsiuria, designating retarded urine excretion, is determined by the opsiuric test. The patient has his supper at 7 P. M., after which he cannot take any more fluid. At 7 A. M. he empties his bladder, after which he imbibes 500 cc. ordinary drinking water, but nothing else (neither to eat or drink) until at 12 o'clock (noon). Punctually at 8, 9, 10, 11 and 12 o'clock the patient is asked to urinate. The amount of urine voided at each micturition is measured. At noon the test is finished., Normally, the urinary excretion increases pretty rapidly until the end of the second hour, after which it decreases rather quickly. Author's observations showed that the opsiuric test is not only of value in the recognition of disturbances in the portal circulation (for the testing of which Gilbert and Lereboullet had especially introduced this method), but that it possesses a certain diagnostic value in other respects. By its employment the motor insufficiency of the stomach may be determined; the method may also be of value in recognizing certain general disturbances of the circulation, particularly in beginning decompensation. It may also be of import in functional renal diagnosis.

WESTERN.

Urinary Findings after Gastric Syphoning—H. POLLITZER and J. MATKO, Wiener med. Wochenschr., July 12, 1915.

Albuminuria supervenes after the stomach is siphoned. The amounts of albumin, however, are smaller than those found by Schiff. The patients in whom an albuminuria supervened after gastric siphoning were affected with disease like status lymphaticus, chronic tonsillitis (some of the latter cases combined with gastric ulcer, tuberculosis or syphilis), goiter, and uric acid diathesis. There was no relationship of the albuminuria to orthostatic albuminuria and habitual lordosis.

MILL.

Tuberculosis of the Male Genital System—M. SIMMONDS, Beiträge z. Klinik d. Tuberkulose, Vol. XXXIII, Nos. 1 to 3.

Genital tuberculosis in the male has a predilection for the third to the sixth, in the female for the first to the fourth decennary of life. In half of the cases it proceeds from the prostate, in a fourth of the cases each from the seminal vesicles and the epididymis. Combined urogenital tuberculosis occurs in the male five times more frequently than in the female. It proceeds most frequently from the kidney, but may originate synchronously in various centers. Hematogenous tuberculosis is histologically not different from the ascending form and, excepting the rare interstitial prostatic tubercles, is due to excretion tuberculosis. Cicatrization processes occur frequently in the testes; in other genital organs they are rare. One-third of the cases of genital tuberculosis die on account of tuberculous meningitis or miliary tuberculosis. Simple castration is not sufficient when there exists a genital tuberculosis. FRY.

Uremia—H. MACHWITZ and M. ROSENBERG, Deutsche med. Wochenschr., Sept. 23, 1915.

Authors have reached the following conclusions: Genuine uremia occurs in acute glomerulonephritis, and in secondary and genuine sclerotic kidney. The diagnosis is most readily made by an examination of the blood for urea or retention nitrogen and indican. Therapeutically, a decrease of albumin in the diet is of value. Eclamptic uremia supervenes especially in the wake of acute nephritis in young people. The most efficient treatment is lumbar puncture. The prognosis is favorable. Atherosclerotic pseudouremia ensues in atherosclerotic affections of the kidneys. It is characterized by the absence of nitrogen retention in the blood and indicanemia.

MILL.

FEMALE ORGANS OF GENERATION—PREGNANCY—PARTURITION—INFANTS

Energometric Observations in the Pregnant, Parturient and Lying-in Woman—W. BIGLER, Zeitschr. f. Geburtshilfe u. Gynäkologie, Vol. LXXVII, Nos. 2 and 3.

Observations on pregnant women at the end of gestation and in non-pregnant women by means of the energometer of Christen. The fulness of the peripheral pulse at the end of gestation is less than normal. This is also the case with its energy and activity. The reason of the diminished peripheral fulness is that the amount of blood necessary for the supply of the genital organs, particularly of the gravid uterus, is withdrawn from the peripheral vessels. The conception that the heart's activity is increased during pregnancy is not contradicted by these facts, because the amount of the increased work which is essential to supply the gravid uterus cannot be measured. It is, however, possible that the extra work of the heart engendered by the gravid uterus is compensated for by a decrease of its activity in the peripheral circulation. The fact that the concurrence of gravidity and heart disease produces decompensation phenomena in so exceedingly few instances speaks in favor of this assumption. The increase reaches its height during parturition. In the puerperium all circulatory phenomena decline materially.

MILL.

Heart Disease and Pregnancy—O. O. FELLNER, Gynäkologische Rundschau, Vol. IX, Nos. 7 and 8.

Author denies that the combination of pregnancy and heart disease is, as such, a serious one. He is of the opinion that one must of necessity differentiate between uncompensated and non-complicated heart affections on the one side and between uncompensated and complicated affections on the other. Women with compensated heart disease get along nicely through pregnancy, labor and the lying-in period. Many instances of compensated heart affections are not brought to the notice of the physician. Generally speaking the physician is only consulted when the pregnant woman has a heart disease in the stage of compensatory disturbances. The heart affection is not serious, but the uncompensated cardiac disturbance is. The otherwise complicated heart disease and the weak heart muscle are also serious when pregnancy is established. MILL.

Functional Determination of Renal Activity during Pregnancy—M. ORLOVIUS, Zeitschr. f. Geburtshilfe u. Gynäkologie, Vol. LXXVII, Nos. 2 and 3.

The determination of renal activity by means of creatinin is a relatively easy matter. The creatinin excretion is retarded when the kidneys are affected. The more retarded the excretion the more extended is the pathological process in the kidneys. Pronounced retardation of the creatinin excretion may be of import in determining the indication for an interruption of pregnancy. MILL.

Genuine Typhlitis and Perityphlitis during Pregnancy—R. T. JASCHKE, Zentralblatt f. Gynäkologie, 1915, No. 37.

Excepting the case reported by author, there is but one of genuine typhlitis stercoralis recorded in literature. A primipara, 26 years old, had 3 weeks before the natural termination of pregnancy an attack of acute appendicitis with high fever. The operation showed a perityphlitic abscess. The child was dead and was removed by anterior hysterotomy. No perforation could be detected on the extirpated appendix. On the other hand, the cecum appeared edematous; it was coated and had assumed a grayish-red color. Symptoms of a peritonitic process beyond the immediate disease region were not present. The patient died from diffuse, suppurative peritonitis. The pus contained the bacterium coli and hemolytic streptococci.
MILL.

A Case of Leukemia during Pregnancy—A. PETERSEN, Archiv f. Gynäkologie, Vol. CIII, No. 2.

Primipara, 24 years old. Spontaneous delivery between the thirty-second and thirty-fourth weeks. Infant alive and healthy. One and a half hour after delivery death of mother in collapse. Necropsy showed leukemia. Association of leukemia and pregnancy is very rare. There appears to be no causative connection between the two. The association of leukemia and pregnancy is of great danger for the patient. Interruption of pregnancy is absolutely indicated.

MILL.

Contracted Pelvis and Difficult Labor—M. HORNSTEIN, Am. Jour. Obstet. and Dis. of Women and Children, 1915, No. 9.

In a woman whose slender build accounts for a small pelvis, small external measurements which are all proportional, with an external conjugate of 15 cm. or over, are not contraindications to a fair test of labor. These cases usually have small children and will deliver spontaneously if the fetus presents favorably. They should be allowed 4 to 6 hours with the membranes ruptured. SACHS.

THE

ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX APRIL, 1916 No. 2

FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York

Published by
REBMAN COMPANY
141, 143 and 145 WEST 36th STREET
NEW YORK, N.Y.

COPYRIGHT 1916 BY REBMAN COMPANY. ALL RIGHTS RESERVED.

Entered as Second-Class Matter, February 8, 1908, at the Post-Office at New York, N. Y.
Under the Act of Congress of March 3, 1879.

Subscription Two Dollars a Year. Single Copies 75 Cents. Foreign $2.50.

THE
ARCHIVES OF DIAGNOSIS
A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX APRIL, 1916 No. 2

Special Articles

INDICATIONS FOR OPERATIVE INTERFERENCE IN DUODENAL ULCER

By JOHN B. DEAVER
Professor of the Practice of Surgery, University of Pennsylvania; Surgeon in Chief, German Hospital
Philadelphia

The indications for operation for chronic duodenal ulcer are symptoms which leave no doubt of the existence of an upper abdominal lesion, causing discomfort with partial or complete disability; morbidity playing a much more important rôle than mortality. When the discomfort is intermittent, as is the case in most of the subjects of chronic duodenal ulcer, the disability is only partial, and the urgency of operation is not so great as when there is complete disability, perforation or hemorrhage.

Perforation is more frequently met with in my experience than is severe hemorrhage; either is a surgical catastrophe and too often the result of medical cures. The strongest language is none too strong in which to protest against the so-called medical treatment of chronic duodenal ulcer, as advocated by many of our gastroenterologists and by a few of our well known internists. I know of no more vicious teaching. These gentlemen can be excused only on the ground of their still being innocent sinners—not having as yet found salvation at the hands of the surgeon, who is making daily excursions through the avenues of the upper abdominal cavity, bringing to the light of day the pathological lesions so frequently found therein, and remedying them with the aseptic scalpel and needle and thread. One prominent medical writer makes the statement that "any ulcer that has not gone deep enough into the mucosa to cause hemorrhage, that has not obstructed the pylorus, or remained chronic long enough to be indurated can be cured medically." This is a bold statement by one who walks by faith and not by sight, a statement to which I take exception. Were it true, the surgeon today would be operating on very few ulcers, as in their life history all ulcers have presented this stage before reaching that of induration, and we take it that the majority, if not all, have been treated medically for many years. I found that the average length of time a duodenal ulcer has been treated is between twelve and fifteen years, and in not a few instances twenty-five years. Statements like the above must influence a great many doctors who, for want of experience in making autopsies *in vivo,* do not know the truth and therefore are spurred on to practice this pernicious teaching, to say nothing of taking advantage of the confidence placed in them by their patients.

In typical cases of chronic duodenal ulcer I rarely have my patients X-rayed, for, even though the X-ray prove negative, I would still advise operation. Although gastric analyses as a rule are of little practical value in the diagnosis of chronic duodenal ulcer, I usually have a gastric analysis made both after a test meal and after a full meal, passing the stomach tube one hour after the former and twelve hours after the latter has been taken; testing the capacity of the stomach after the removal of the test meal, if there are no contraindications to this, by filling it with water, removing and measuring the same and inflating it with air, in order by percussion to determine its position. In the diagnosis of gastroptosis I rely more upon the above procedure than upon X-ray findings, believing it to be a more certain method for demonstrating a gastroptosis than the bismuth meal and the X-ray. There is no doubt that the weight of the bismuth meal and the erect posture are factors in aggravating the degree of gastroptosis. Furthermore, it is my experience, that the ordinary case of gastroptosis can be diagnosed by palpation, auscultation and percussion, and by noting any marked pulsation of the abdominal aorta, as well as by the history of obstinate constipation.

In only a small percentage of patients does the gastric analysis in typical cases show a very high acidity. Hyperchlorhydria is a feature of cholelithiasis, gastric ulcer, colitis, tuberculosis of the intestine, tuberculosis of the mesenteric gland and appendicitis. The history alone suffices to make the diagnosis in typical cases of duodenal ulcer, the symptoms, in my judgment, being sufficient basis to serve as an indication for operative interference. The diagnosis of pyloric obstruction with food retention and dilatation of the stomach, acid eructations with, in some instances, vomiting is readily made. Röntgen examination can only confirm that which has already been proven.

The first symptom in duodenal ulcer is usually a sense of weight, oppression or distension in the epigastrium after eating. Distension is sometimes so pronounced that the patient will speak of it as a feeling as though the abdomen were going to burst. The pain and discomfort are relieved by eating. The pain following three or more hours after eating is first noticed after the heaviest meal of the day, but later appears after all meals and is described as boring, gnawing and burning; at first gradual and slight, it soon increases in severity and is accompanied by a feeling of fullness, which is relieved by belching. Pain appearing on an empty stomach when the patient commences to be hungry suggested to Moynihan the name of "hunger pain."

In the last twenty-eight cases of chronic duodenal ulcer operated on by me only five had a hyperacidity. The presence of occult blood in the stomach washings is of no importance, nor do I regard the string test and the use of the duodenal bucket of any use except for the impression they may make upon the patient, who, if at all intelligent, will attach but little importance to so trifling and disagreeable a procedure. Personally, I consider these tests merely playing at an attempt to make a diagnosis. High acidity or subacidity is typical of chronic duodenal ulcer in a percentage of cases. In my experience it is in the active stage of ulceration that an excess of hydrochloric acid is most often found, being present in the late stages only when there is stenosis and dilatation. It is important to know the amount of excess of hydrochloric acid in the stomach contents, particularly in the early stages of the disease.

Occult blood in the feces as ordinarily tested for, with no regard to the previous diet of the patient, counts for nothing, although important if found daily after a meat-free diet. It is usually present in the acute stage of ulceration. It must be remembered that there are many other conditions of the alimentary canal that give occult blood in the feces besides chronic gastric and duodenal ulcers, such as gallstone disease, pancreatitis, etc. Macroscopic blood in the feces is, in my experience, rare. Hemorrhage in duodenal ulcer either by way of the mouth or the rectum I have seen in a limited number of cases only. It is therefore not a sign of great diagnostic value in the early stages of an ulcer. In atypical cases, where the diagnosis is uncertain, I advise X-ray examination more for the purpose of aiding the diagnosis than in the expectation of definitely establishing it. It is my opinion that X-ray examination (which of course includes fluroscopic examination) is reliable and to be considered only when made by experts as Pfahler, Pancoast or Cole.

Indigestion as a symptom is as aimless and fruitless as traveling in different parts of Europe without a guide to interpret the different languages spoken by the various nations. In order to interpret the term indigestion it is necessary to familiarize oneself with the different languages spoken by the different portions of the alimentary canal, and it is only natural that the surgeon who has opened the abdomen the greatest number of times is best equipped for the purpose and therefore the best guide for the medical man.

The duodenal ulcer patient may doubtless be temporarily relieved by observing precautions as to diet and by frequent doses of alkalies. This is plainly shown by the histories obtained. At the same time such an individual who, having been warned of the danger of not having an operation, prefers to have medical treatment, takes this risk on his own responsibility.

The indications for operation for chronic duodenal ulcer, I repeat, are those of an unquestionable upper abdominal lesion. Conditions most likely to be confounded with chronic duodenal ulcer are chronic appendicitis, cholelithiasis, chronic pancreatitis (chronic pancreatic lymphangitis and chronic interstitial pancreatitis) and chronic gastric ulcer near the pylorus. It has been well said by Moynihan that the most common location of gastric and duodenal ulcer is in the appendix.

The chief points in the differentiation between a chronic appendicitis that mimics the symptoms of chronic duodenal ulcer are that the indigestion of the former is constant, persistent and unremitting, not varying as in ulcer or cholelithiasis; that certain articles of food such as meat and starchy substances aggravate the symptoms, and that in addition to constipation the distress comes on immediately or soon after eating and is accompanied by nausea or vomiting. The relation of the pain with ingestion of food sometimes corresponds to that of ulcer, and when associated with hyperacidity, often present in appendicitis, may suggest chronic duodenal ulcer. In chronic appendicitis constipation is the rule, and let me add, it is the *result* and *not* the cause of the appendicitis. More or less intestinal distension due to an excessive amount of gas is also often present and percussion will frequently reveal a slight distension of the abdomen of these patients.

The presence of tenderness with some degree of rigidity of the overlying muscles is an important differential point. The site of the tenderness and rigidity will depend upon the location of the appendix. The right iliac fossa is the most common location of the appendix, although in quite a number of patients it lies to the outer side of the cecum and the ascending colon and sometimes behind the colon, pointing upwards. The necessary pressure to elicit tenderness of the appendix will often cause the same kind of pain the patient complains of, and refers to the epigastrium or to the right lower chest margin. Pain or discomfort in chronic appendicitis is frequently made worse by exercise. Patients suffering from chronic appendicitis will occasionally show occult blood in the stools.

Pain one hour after eating indicates gastric ulcer and when occurring two hours after eating the ulcer is near the pylorus. Unlike duodenal ulcer, gastric ulcer is not affected by seasonable change. In gastric ulcer if the pain is not confined to the epigastrium it is usually referred to the lower right or left chest margin. Pain in the back indicates pancreatic involvement; vomiting of blood, gastric ulcer.

In cholelithiasis the pain is much more severe; it is usually referred to the right shoulder blade and is accompanied by nausea and vomiting soon after eating, with belching of gas and hepatic colic appearing and disappearing with equal suddenness. This is a common disease of the Jews, and particularly the female, who is fair, fat and forty, and belches gas.

A carefully elicited history, not only in appendicitis, but in the other conditions mentioned, is the most important step in the differential diagnosis. In chronic duodenal ulcer the history shows that the patient has in many instances suffered all his life, that the symptoms, referred to the stomach, appear insidiously in the shape of weight or oppression, or epigastric distension after meals. Pain or discomfort before eating is usually relieved by eating. When the pain appears comparatively early after eating, say, one hour or a little longer, it is suggestive of adhesions of the ulcer to neighboring parts or of an incipient obstruction. Liquid food is followed by pain earlier than when solid food is taken. As long as the patient confines himself to one kind of food the pain appears at the same time daily, but any decided change in the character of the meals influences the time relation of the subsequent pain. Vomiting may occur without affecting the appetite. The chief point in the symptomatology is the periodicity of the attacks of pain. The last patient I have operated, 57 years old, has had the symptoms for 25 years, three or four attacks yearly, starting with pain only when the stomach was empty.

The effect of cold as an exciting cause of an attack is evidenced by the fact that we see more cases in winter than during the warmer seasons. Between attacks the patient is perfectly well, and the medical treatment is credited with a cure. Physical examination, as a rule, except when made during an attack of pain, when tenderness and rigidity can be elicited, avails very little. Blood evacuated by mouth or bowel is an indication of a late stage of the disease. The ulcer that has invaded the deeper tissues of the bowel, means that the golden opportunity of doing an easy and safe operation is passed; that the medical treatment has utterly failed.

Hemorrhage is not a symptom but a late complication, for which someone is responsible. Who? In my experience hemorrhage in duodenal ulcer is far more dangerous than is hemorrhage in gastric ulcer, where it is rarely fatal, while it does kill in duodenal ulcer. Bleeding from an eroded vessel in a chronic duodenal ulcer, let me repeat, is a grave symptom, as the vessel can neither retract or contract.

Perforation in acute ulcer I have never seen, or have failed to recognize it if present. The diagnosis of acute perforation is not difficult. With few exceptions, in the large number of hospital cases I have operated, the diagnosis has been made by the house surgeon before I have seen the patient. In practically all of them a history of chronic duodenal ulcer can be elicited; the exception being when the patient cannot speak our language and an interpreter is not at hand. The pain is most acute and agonizing, second in importance and severity to none, and is immediately followed by a rigidity of the abdominal walls familiarly characteristic to the experienced observer. I tell my students that the rigidity is so pronounced that one could stand on the abdominal wall without in any way causing it to yield. This rigidity is accompanied by a typically fixed position of the patient, who resists any attempt at changing it. Tenderness is, if anything, more evident over the upper right abdomen. This of itself suffices to make a diagnosis spelling immediate operation. I have never seen a case of shock in my experience, numbering over forty-four cases, in dealing with this catastrophe. These cases were all operated and all recovered, with one exception (a man sick four days, having a general purulent peritonitis). The operation in all of the cases, with two exceptions (the fatal one being one of the two) consisted of closing the perforation, plication of the duodenum, posterior gastroenterostomy and removal of the appendix.

I know that many surgeons take exception to doing a posterior gastroenterostomy as well as to the removal of the appendix. I maintain my position is a sound one. For the expert surgeon I claim this is the correct treatment; the occasional operator had better content himself with simple closure of the perforation. Simply to introduce drainage and to do nothing else is to hand the case over to the Great Physician. If the patient recovers, as I have known to occur, the Great Physician has been kind to the operator, but especially so to the patient.

I have seen and operated a few cases of sub-acute perforation, a condition not so infrequent as is generally supposed. It differs from acute perforation only in that the local process is a limited, circumscribed one. Here again the history makes the diagnosis, and, as in acute perforation, constitutes the indication for operation.

THE VALUE OF DEEP PERCUSSION IN THE DIAGNOSIS OF SUBACUTE INTRAABDOMINAL DISEASE

By HAROLD NEUHOF
Surgeon, Mount Sinai Hospital Clinic; Hebrew Infant Asylum; Sanitarium
for Hebrew Children; Associate Surgeon, Har Moriah Hospital;
Assistant Neurological Surgeon, Central and Neurological
Hospitals, Neurological Institute, and
Montefiore Home
New York

In patients who first come under observation after acute abdominal symptoms have largely subsided, and in patients presenting the symptoms and physical signs of a subacute intraabdominal affection, the diagnosis can be made readily in many instances. There may be a painful mass, or an area of sharply localized pressure-tenderness, a zone of cutaneous hyperalgesia, a history typical of the affected organ in the absence of clear physical signs, etc. However, in a fair proportion of the cases under consideration, and especially in the obese, it is often difficult and not infrequently impossible to locate the seat of disease by the usual methods of physical examination. It was for these cases that the value of deep percussion as an aid in diagnosis was first pointed out about four years ago.* Since that time instances of its usefulness have multiplied to such an extent that the method has, in my hands, become part of the routine examination for the types of abdominal affections in question. It is logical to suppose that deep percussion is employed by others, perhaps as a routine, but a description of its practice is not mentioned in the literature.† The purpose of the present communication is therefore a desire to call attention anew to the method, on the basis of additional experience.

Two forms of percussion are used, the object being to elicit localized tenderness. One is the application to the abdominal wall of the ordinary deep percussion as employed in chest examination. A moderate amount of rebound force may be required to disclose the painful area. When this fails, or the result is doubtful, the second method is used. It consists in tapping the abdominal wall with the crooked finger of right hand. For evident reasons it should be emphasized that these methods of deep percussion can be employed only after the history and physical examination (always including rectal or vaginal examination) clearly indicate that the patient is not suffering from an acute abdominal condition.

---

* H. Neuhof, Medical Review of Reviews, June, 1912.

† The only other described method of percussion, employed to elicit tenderness, is the light ("piano") percussion used by J. B. Murphy in the diagnosis of acute abdominal conditions.

The value of deep percussion in subacute or largely subsided intraperitoneal affections rests in the fact that tenderness is elicited when deep palpation may not disclose any specially painful area. I have found well-localized sites of deep percussion-tenderness in the complete absence of pain on deep pressure. Almost invariably percussion-tenderness is definitely circumscribed in a small area of the abdomen over the diseased organ. Pain induced by deep percussion is usually mild, occasionally moderate; with the proper selection of cases it should never be severe. In a number of instances it was the only physical sign that led to the correct diagnosis, as I was enabled to prove by operation. It was also demonstrated by many operations that percussion-tenderness is not a misleading physical sign. The diseased organ was usually found in the immediate neighborhood of the painful area, and a diseased organ was invariably encountered when percussion-tenderness had been present. It should be stated, however, that the extent of pain induced by deep percussion is no index of the severity of the lesion found at operation. Not infrequently I have been surprised by the considerable tenderness in patients suffering from mild subacute and mild subsiding lesions.

It is not necessary to recount the individual cases in which percussion-tenderness has been of definite value in the diagnosis. The type in which it has been of greatest use is the very obese individual who first comes under observation with the history of an acute abdominal condition that existed a few days before. Routine physical examination reveals nothing besides vague soreness of the abdomen that cannot be definitely located by the patient or by the examiner. Deep percussion induces well-defined tenderness over the affected organ. It is of interest to note that percussion-tenderness is very readily obtained when the abdominal wall is rigidly held. This is an important practical advantage of deep percussion over deep palpation employed to elicit localized tenderness. At the same time it suggests an explanation for the percussion phenomenon: Deep percussion of the abdominal wall sets in motion waves that finally impinge on the diseased viscus. On the basis of physical laws it is evident that rigidly held abdominal parieties would offer no bar and, indeed, might aid in the evolution of such waves. Without further discussion of this point it may be said that, theoretically, deep percussion should aid in locating a diseased intraabdominal focus, regardless of whether it was in or around one of the abdominal viscera. In practice, however, the method is confined to subsiding or subacute infections, so that this question could not be satisfactorily determined.

In conclusion: Deep percussion, as a means of eliciting localized tenderness, has proven of great value in the diagnosis of subacute and subsiding intraabdominal affections, especially in the examination of obese individuals and of those who do not sufficiently relax their abdominal wall for satisfactory palpation. The method is free from danger and should therefore be made part of the routine physical examination in subacute and subsiding intraabdominal affections.

THE ETIOLOGY AND DIAGNOSIS OF INFECTIVE DISEASES OF THE BILIARY DUCTS

By G. A. FRIEDMAN
Visiting Physician Beth David Hospital; Attending Physician in Digestive Diseases Vanderbilt Clinic; Gastroenterologist
Gouverneur Hospital
New York

The commonest cause of infection of the biliary ducts is an infection of the reservoir for bile, the gall-bladder. From any primary focus of infection in that organ, no matter how mild, the common, the cystic, the hepatic and the intrahepatic ducts may become involved, resulting in cholangitis, angiocholitis, acute or chronic, catarrhal or suppurative.

With a very mild infection in the gall-bladder, the infection of the biliary passages may remain latent. In an infected bladder, stones may or may not be present. Experiments have proven conclusively that the colon and the typhoid bacillus produce cholecystitis which is followed by the formation of calculi. Although nonvirulent cultures of streptococci and staphylococci may also give rise to calculi, yet virulent cultures of these organisms set up an intense cholecystitis which may be followed by cholangitis without the production of stones. It has been shown lately by Rosenow, that cultures from the tissue of an actively diseased gall-bladder injected into the veins of animals produced disease of the gall-bladder by selection, and occasionally even stones. But whether stones are present or not, the frequent association of infected gall-bladder and infected bile passages is evident. Inflammation of the bile ducts is greatly enhanced by the presence of calculi in an infected gall-bladder. It has almost become a general belief, that a mild infection of the gall-bladder precedes the formation of calculi, or in other words, cholelithiasis is the sequel to cholecystitis, not cholecystitis to cholelithiasis. But this must be modified in so far that, while gall-stones are not the chief cause for cholecystitis, the continued presence of calculi in the gall-bladder facilitates the development of a mild or an attenuated infection to a moderately severe one. This infection may then readily spread to the larger ducts. According to the statistics of Rogers gall stones were found in 18 out of 20 cases of suppurative cholangitis.

The mildest cholecystitis may lead to calculi, for by its presence the metabolism in the secreting cells of the mucous membrane of the gall-bladder becomes disturbed, with the result that cholesterin is formed in these cells. Stones that are found in the larger bile ducts have not formed there, but have become wedged in these ducts on their way from the gall-bladder to the intestines. When, however, as in exceptional cases, calculi are formed in the ducts, an infection, even though very mild, must have preceded their formation. This infection leads, however, to an exudation of albuminous material which precipitates bilirubin in chemical combination with calcium, bilirubin calcium calculi. But in case of an impacted stone in the common duct, calculous material containing cholesterin may also be formed. The presence of biliary calculi may not show clinically any signs of infection.

A third cause for infection of the biliary passages is stasis of bile either in the bile ducts or in the gall-bladder. It is a well known fact that in the normal state, the bile, the gall-bladder, and the biliary ducts are aseptic, the only exception being the lower portion of the common duct in which the coli bacilli, streptococci, and staphylococci may be normally present. Sometimes the pneumococci, the cholera bacilli, and liquifying saphrogenic bacilli, and in patients who had typhoid fever, the typhoid bacilli may be present. There is a strong evidence to warrant the belief that the terminal portion of the duct is the first to be involved.

The question at this point, why should the organisms suddenly become pathogenic in some individuals and remain harmless in others presents itself. It is probable that an increase in the virulence of the intestinal microbes may be sufficient cause for their emigration to the biliary passages. But the assumption that there is a change in the mucous membrane of these ducts, or a change in the state of the bile, or in both, seems more plausible. A change in the mucous membrane nearly always involves a change in the bile, so that there may be an interference with its flow, leading to stasis, or it may lose its aseptic properties and become infected, forming a favorable medium for bacterial growth.

The pathological factors which involve the mucous membrane are many. Among the more important ones my be mentioned; first, partial obstruction and injury due to calculi from the gall-bladder or exceptionally from the larger ducts; secondly, a gastroduodenitis from any cause, either from indiscretions in diet, alcoholism, repeated chills, exposure, or chronic heart or renal disease; gastroduodenitis producing a catarrhal condition of the papilla of Vater which in addition, may become plugged by a piece of tenacious mucous, causing a swelling of the mucosa of the biliary passages and the retention of bile in the intra-and extra-hepatic ducts and preparing the way for a bacterial invasion; thirdly, by pressure of tumors of these and neighboring structures; and finally the occasional occurrence of foreign bodies becoming wedged in the common duct. The result is the same—a swelling of the mucosa of the biliary passages, a stagnation of bile and *the infection.*

The following will illustrate that something more than bacteria is necessary to bring about an infection of the biliary passages. A boy of twelve, who was under the writer's observation several months ago, and whose past and family history are practically negative, complained for five days of indigestion. On the sixth day, he was seized with chills, fever (103.5 deg. F.) and jaundice set in.

His liver was slightly enlarged, his spleen, markedly enlarged and tender. The fever being of the intermittent type, was present for three days. His urine was brown, his stools, clay-colored. A leukocytosis (12.600) was present. A search for plasmodiae malaria proved negative. On the 12th day of his illness, the jaundice disappeared entirely; several days later his stool and urine assumed the normal color. This was, I believe, a case of catarrhal jaundice. There seems to be no doubt that in many cases of "so-called catarrhal jaundice," the infection of the biliary passages comes from the duodenal catarrh. For instance, in this boy, the gastroduodenitis which preceded the fever caused a swelling of the mucous membrane of the duodenum, together with that of the papilla of Vater, which requires comparatively little swelling to obstruct its lumen.

With the cattarrhal condition there is produced an excessive amount of mucus which may be another factor in the more or less partial obstruction of the lumen. The swelling of the mucous membrane may extend to the larger and to the smaller ducts. The consequence is, as stated before, a stasis of bile. The next step is the extension and passage of the organisms into the ducts, and their growth in the tissues and stagnant bile. If pyogenic organisms are the invading bacteria, suppuration may result.

Stasis of bile in the biliary tract may be produced by the same factors which predispose to stagnation of bile in the gall-bladder. Sedentary life and want of exercise, which lead frequently to feeble contractions of the abdominal muscles, favor stasis of bile, and as a result, bile is not expelled as frequently or as efficiently from the gall-bladder. Tight lacing of corsets may lead to dilatation of the gall-bladder from the duodenal straining and cause kinking of the cystic duct. A displacement of the right lobe of the liver may also kink the cystic duct, for the fundus of the gall-bladder then becomes more dependent than in the normal state, the cystic duct remaining fixed in the transverse fissure. A nephroptosis to the right favors stagnation produced by traction on the peritoneum covering the common duct.

Impediment of diaphragmatic respiration is apt to cause stasis in the biliary passages by interfering with the proper emptying of the gall-bladder. Among the conditions causing difficulty in diaphragmatic respiration, we may number tight lacing, pregnancy, ascites, abdominal and thoracic tumors, and pulmonary and cardiac diseases.

Among the rarer factors leading to infection of the biliary passages, the following deserve special attention. Pieces from a hydatid membrane, like that from a ruptured echinococcus cyst of the liver, may be the cause. The writer lately had the opportunity to observe such a case at the Gouverneur Hospital. A Greek boy of 18 was suddenly seized with excruciating pain in the upper abdomen several days before his admission to the hospital. Jaundice accompanied the onset. During the first five days of his stay in the ward, he had a septic temperature and a marked leukocytosis. The liver was not enlarged. He had no cough and no signs of pleural irritation. His urine gave repeatedly a positive reaction for bile and urobilin. The stools were of normal color, contained bile, and the quantitive estimation of pancreatic ferments was low. His past history was negative except that five years previous to the present attack of pain he had a similar one in the upper abdomen but unaccompanied by jaundice.

A diagnosis of pancreatic abscess and cholangitis was made. At the operation performed by Dr. Ladinsky, a small abcess was found in the head of the pancreas, and a hydatid cyst on the upper surface of the liver posteriorly. Subsequently, it was elicited that the pathologist of the hospital, Dr. Schwartz, found, at one examination, ova in the feces. The ruptured cyst had probably led to the infective cholangitis and the formation of pus in the pancreas.

Again, in rare instances, roundworms may pass up the common bile duct from the duodenum and carry microorganisms with them, or cause changes in the mucous membrane. In primary abscess of the liver, the inflammation is liable to spread to the bile ducts and produce a secondary cholangitis. Carcinoma arising on the duodenal surface of the biliary papilla, or carcinoma of the diverticulum of Vater and of the lower portion of the common duct may also lead to infection of the bile passages.

Appendicitis is not infrequently associated with cholecystitis, and, in the writer's experience, with angiocholitis. On several occasions he has found a strong reaction for bile in the urine in patients in whom appendicitis was found at operation. In these cases, stones in the gall-bladder were not present; there was absolutely no evidence of an inflamed gall-bladder.

A boy of 18 suffered from attacks of biliary colic without any tenderness in the appendicular region. His urine, which had been repeatedly examined, showed a strong reaction for biliary pigments. The only reason for diagnosing the case as appendicitis in the absence of focal symptoms was the age of the patient, for gall-bladder disease is not common at such an early period of life. Somewhat later the patient was operated upon by Dr. Silver, and I learned that he found a chronic appendicitis with no inflammation or calculi in the gall-bladder. Therefore, an infected condition of the intrahepatic ducts due to the inflammation of the appendix may have been responsible for the persistent presence of bile in the urine.

The inciting agents are microorganisms. The varieties of bacteria which have been found are numerous. The great tendency of some bacilli, especially the colon and typhoid, to invade the gall-bladder and the ducts, can hardly be disputed. The colon bacilli, the streptococci, and staphylococci are constantly present in the intestines, and under pathologic conditions, any organism may become an inhabitant of that tract, and travel to the bile ducts. There are several pathways by which these bacteria may reach the biliary passages. It has been shown that in some cases the bacterial infection from the duodenum occurs by way of the diverticulum of Vater and the common bile duct. It is stated that the colon bacillus takes this route more frequently than the typhoid bacillus. The typhoid bacillus seems to prefer the portal circulation in reaching the biliary passages from the intestines. Normally bacteria carried to the liver by the portal circulation are destroyed by the bactericidal properties of the liver cells. When, for some of the reasons mentioned, this function of the liver cannot be properly performed, infection of the biliary passages may result.

A third and a less common mode of infection is the general circulation. This is seen in the acute infections such as pneumonia, influenza, typhoid and sepsis. The occurrence of jaundice in pneumonia, though rare, is well-known. The writer remembers having spared a colleague from operation by recalling the fact that the attacks of abdominal colic, rigors, fever, and jaundice followed a severe attack of grippe from which he suffered two months previous to the present appearance of these symptoms. In these specific fevers the infection seems to be a true hematogenous one, rather than an acute gastritis with its accompanying duodenitis.

Lately it has become known that infection of the biliary passages may also occur through the lymphatics by direct extension. This may explain the cholangitis accompanying appendicitis.

Since several affections of the biliary passages, as cholecystitis, cholelithiasis, and cholangitis may be associated, it will frequently be impossible to make a positive diagnosis of the condition confronting us. In such cases, a diagnosis of biliary infection should be made without attempting to ascribe the symptoms or signs to the gall-bladder or to the extra- or intra-hepatic ducts. In addition, it will frequently be impossible to state whether stones are present in the gall-bladder.

Rigors, intermittent fever and sweats may be expected in any case of moderately severe or severe infection. However, attacks of chills, fever, and sweats by no means warrant a diagnosis of suppuration of the biliary tract, though these are evidence of infection, but not necessarily of pyogenic infection.

Attacks of jaundice are liable to occur in cases in which there is partial obstruction, either due to stones or to a swelling of the mucosa. But in complete obstruction jaundice is marked and persistent, while chills, hepatic fever, and sweats may be, and usually are absent. We may expect catarrhal or suppurative cholangitis to be present in patients suffering from attacks of chills, fever, and sweats, whose past history points to gall-stone colic, or who have had typhoid fever. The assumption of cholangitis should not be dismissed even if jaundice is not pronounced or even if it be absent. The finding in such instances of hepatic or splenic enlargement or of enlarged gall-bladder with a polymorphonuclear leukocytosis will place the diagnosis of cholangitis beyond any doubt.

In a number of cases of infective diseases of the biliary passages, the history and the symptoms are vague: a clear history of gall-stone colic cannot be elicited. We may encounter the following conditions: Attacks of chills, fever, and sweats, but malaria in such cases will have to be excluded when there are no plasmodia in the blood. Pylephlebitis, which may give similar symptoms, and which is a complication of hepatic abscess, should be ruled out by the absence of antecedent dysentery. Suppurative cholangitis, even if jaundice be absent, then becomes a possibility. To confirm the diagnosis, there are no better signs, in the experience of the writer, than the finding of tender spots in the region of the gall-bladder, in the axillary and in the scapulary lines on the right. Those spots, when carefully searched for, will be found almost in every patient with cholelithiasis, whether he had typical attacks or not. These spots frequently tell a much clearer story than the patient.

The following case may serve as an illustration of this statement:

A housewife of 42, first seen by me on March 27, 1913, gave a history of having been ill for one year with attacks of epigastric pain, coming on from one-half to two hours after meals, and radiating to the left. She had never had any pain on the right side, had vomited on several occasions, and was jaundiced for one day at the onset of her illness. She never had any chills. Her past history was negative. She refused to be examined by means of the stomach tube. Nevertheless, a tentative diagnosis of gastric ulcer was made, as her history, with the exception of one day jaundice, pointed distinctly to this condition. It must be observed here that many patients, when speaking of jaundice, mean pallor. The patient was put under ulcus therapy after which she remained well until May 1, 1913. On this day she had an attack of what she called "nervous chill," but did not suffer the slightest pain. Her temperature was 103 by mouth. Leukocytosis 16,000 of which 89 per cent. were polymorphonuclear leukocytes. On this day the diagnosis was made certain, though suppurative cholangitis had not been at first suspected. As there was no etiological factor in her history to account for the suppurative condition, the presence of the tender spots in the right axillary and scapulary lines was attributed to cholelithiasis which previously had given no acute symptoms. Several hours after the onset, an operation was performed by A. A. Berg. A large number of stones were found in the gall-bladder, and also calculi in the cystic, hepatic, and common duct. The common duct was filled with pus and adhesions between the gall-bladder and duodenum, and a fistula between gall-bladder and stomach was present.

Attacks of fever, chills, and sweats in an elderly person who is cachectic and icteric, will occasionally lead to the diagnosis of cholangitis, which may be due to carcinoma of the papilla of Vater or to carcinoma of the lower end of the common duct. Occasionally it will be necessary in a case of cholangitis with obscure etiology to search for ova of taenia echinococcus, as a hydatid cyst may develop on the convexity of the liver without any symptoms or signs. The aforementioned case illustrates this eventuality.

In recent years, X-ray exposures revealed shadows which point to gall-stones. These findings depend upon the experience of the radiologist. Indirectly, they may solve the etiology of the infection in obscure cases.

BANDS AND ADHESIONS IN THE CECAL REGION

By HERBERT E. STEIN
Assistant Gynecologist, New York Polyclinic; Chief, Surgical Clinic, Harlem Hospital
New York

In every scientific subject that is of recent origin or of reawakened interest, we find the literature replete with innumerable theories, especially relating to the etiological phase. These numerous views result in an inevitable chaos until one unifying thought satisfies the various ideas entertained by all the investigators. In the matter of the so-called Jackson membrane and Lane's kink we have just about arrived at a plausible solution of the etiological problem. To whom priority is due in first calling attention to this subject there is still doubt. According to Hofmeister, Virchow first described the condition Jonesco, Juvara, Treves and Reid all called attention to the bands, though under a different nomenclature from the one now in use. Binnie and Lane undoubtedly recognized them. To Wilms we are indebted for having directed attention more closely to cecal conditions, and to Jackson and Lane belong credit for accurately describing the membranes that are known by their names and for possible pathological results these bands may produce. A source of confusion has been due to the failure of making a pathological examination of the membranes removed. Undoubtedly, not a few so-called Jacksonian membranes have been true adhesions and thus the cause been made to fit the result. We will consider the Jacksonian membranes and Lane kinks separately, although they are probably due to one and the same process.

Etiology.—The presence of the bands has been ascribed to different causes, which we may classify as (1) congenital; (2) acquired.

Congenital. (a) Various observers have seen in the fetus what corresponds to the adult type of Jacksonian membrane. A parieto-colic fold was found by Jonesco and Juvara in twenty per cent. of the fetus older than 6 months. Eastman found it present in five out of twenty-eight, Cheever in four out of thirty still-births, and Reid observed it in three out of twenty fetuses, *i. e.*, from twelve to twenty per cent. Flint reports several cases, two having been infants at term. To account for these bands in embryo we must remember the three distinct changes which the cecum undergoes in its development, *i. e.*, migration from the left iliac region upwards and across the abdomen to the hepatic region where rotation takes place, followed by descent to the right iliac region. The fixation normally takes place between the opposed surface of the cecum, ascending colon and parietal peritoneum antoriorily to the right kidney. If fusion takes place before descent of the cecum, the latter in its descent will pull the fine peritoneal bands down with it in an oblique direction. Fetuses at different ages show bands of different lengths, depending on the extent of the cecal descent. If fusion occurs before rotation is completed, the peritoneal fusion will occur between the anterior and internal aspect of the cecum or colon and parietal peritoneum. Thus, as rotation progresses, the veil will be seen extending across the front and internal surface of the gut. In both these types the fusion may occur directly between opposed peritoneal surfaces or one or more of the appendices epiploicæ may fuse with the pre-renal parietal peritoneum and thus draw out the connection as the cecum descends. The membrane extending from the antero-internal aspect of the cecum and ascending colon to the omentum may likewise be attributed to an atypical fusion of the posterior meso-gastrium, which is the place of attachment of the omentum, and the cecum while still in its sub-hepatic position. When the lateral parietal peritoneum, the visceral peritoneum and the omentum fuse, the veil extends from the omentum across the anterior aspect of the colon to the lateral abdominal wall.

(b) Mayo has advanced the theory that the cecum, in its descent burrows its way through the parietal peritoneum, which had been developed before the cecum began to descend. This is comparable to the descent of the intestine in the peritoneal sac, as occurs in a hernia. Just why the cecum should take this difficult route is rather problematical. Furthermore, if Mayo's theory is correct, it would appear as if the cecum and the whole ascending colon ought to be enveloped in or at least covered by a membrane. Such a condition is only rarely encountered in one and the same patient.

2. Acquired. (a) The formation of the parieto-colic membrane as well as Lane's kink may be due to an attempt on the part of Nature to overcome the effects of gravity. The evolutional changes man undergoes from the horizontal to the erect position all tend to a sagging or prolapse of the viscera (Martin). In the lower animals the peritoneal attachments within the abdomen are very loose and the lateral abdominal wall, especially of quadrupeds, consists of strong fascia. In man, the mesenteries are short and the fascial wall is substituted by strong abdominal muscles. In quadrupeds the sacrum makes a straight line with the rest of the spinal column, whereas in man the pelvis gradually assumes a position almost at right angles to the rest of the spine, which takes on a compensatory cervical and lumbar curvature. This tends to protect the pelvic organs from intra-abdominal pressure, which is diverted toward the anterior abdominal wall. Defective nutrition, emaciation, flabby abdominal muscles, loose peritoneal attachment of the intestine, and straight line formed by the sacrum and spinal column tend to a prolapse, and excessive mobility of the abdominal organs and intestinal stasis. This evolutionary defect Nature attempts to overcome by the formation of new bands and adhesions. This theory may be applicable to a few cases. But the fact that the bands and membranes have been demonstrated in only a small percentage of the embryos examined for these veils, and as the development of the fetus is but a résumé of the evolution of man, we conclude that the evolutionary theory should be discarded. (b) Inflammatory colitis or repeated mild infections of the colon with the resulting fermentation, putrefaction and stasis in the cecum and colon have been considered the cause of the pericecal and pericolic membranes. Binnie, Gerster and Pilcher entertain this view, claiming that the bacteria are capable of passing through the wall of the intestine, even without the presence of any ulcer.

Pathological conditions of neighboring organs such as the duodenum, appendix or gall-bladder have been held responsible for the development of the membrane by Hertzler and Hofmeister. The former has called the condition "varicosity of the peritoneum," due to a more or less distant seat of inflammation. Gerster states "exosmosis of the bacteria through the wall of the intestine leads to denudation of its endothelial covering, which may result in adhesions of adjacent surfaces." Further on in his articles he contradicts himself by stating that these membranes about the cecum and colon differ from ordinary adhesions in that they "do not enter into a close organic union even with the serous coating of the gut. They can be stripped off without difficulty and after their removal the intact and glistening serous coat of the intestine is exposed. True, they do adhere, but in a very loose manner." If not a contradiction, this is the description of two different pathological entities, *i. e.,* a true adhesion and a parieto-colic membrane. Again he states: "Their (the membranes) attachment to the parietal peritoneum, evidently represents the oldest part of the growth. This is evidenced by the presence of a narrow, glistening, fibrous band of tissue, which runs parallel with the longitudinal axis of the gut and assumes a scalloped outline whenever the gut is pulled inward." Here again it would seem that the oldest part of any adhesion would most rationally appear at the source of irritation, which, according to Gerster's view, is the colon and not the parietal peritoneum. "Their substance," according to the same author, "is a transparent, myxoma-like tissue." This would be rather unusual evidence of an inflammation. From these various data the inflammatory theory as to the origin of these membranes is hardly tenable. The loose attachment of the membrane to the colon, the absence of or slight bleeding on its removal, leaving a normal peritoneal coat, the transparency, vascularity, more or less regular distribution and no thickening of the wall of the gut, rather speak against inflammatory manifestations. Furthermore, microscopical examination does not show any fibrin, leukocytes, or, in fact, any inflammatory condition, according to Hall. The endothelial lined spaces are enlarged, suggesting a lymph stasis. The peritoneal structure is only thinned out.

It appears to us that the most plausible explanation of these much discussed membranes is that they are the result of an atypical fusion in the fetal life of the cecum and colon, together with the parietal peritoneum or omentum, and that for some reason they are not absorbed but persist. This condition might be comparable to an overproduction of callus with the persistence of an excessive permanent bone formation.

In case of ulcerative typhlitis or colitis, there is no reason why adhesions should not form about the area involved, just as they do about a gastric or duodenal ulcer. But these are true adhesions and quite easily distinguished from so-called Jackson membranes.

Pathology.—The membrane is, as a rule, a very delicate, glistening, transparent veil, containing long, straight, parallel, non-anastomosing blood vessels which are probably derived from vessels of the peritoneum and terminate in those of the intestine. The blood vessels are accompanied by narrow connecting tissue bands. The general direction of the veil is from the external abdominal wall downward and inward, or from the transverse colon and upper omentum downward and outward, running spirally partly around the gut. At its attachment to the intestine the membrane usually spreads out fan-shaped and causes a plication of the gut wall. The membrane is considerably thinner than the parietal peritoneum. The large intestine is usually freely movable under and non-adherent to the membrane, nor has the latter been reported adherent to loops of small intestine or to the anterior abdominal wall.

There are two types of parieto-colic membranes as far as location is concerned, though the minor variations are quite numerous. One extends from the lateral abdominal wall to the anterior and lateral aspect of the ascending colon or cecum, or both; the other unites the omentum and the first portion of the large intestine. The first type may involve the hepatic flexure and ascending colon and terminate at the cecum, leaving the latter and the appendix free. The shorter veil starts lower down on the parietal peritoneum, involves the lower portion of the ascending colon, the whole cecum and the proximal portion of the appendix, which becomes sharply kinked. Occasionally the whole appendix is enveloped. The cecum frequently becomes dilated and thin-walled. The second type involving the omentum may extend across the hepatic flexure and upper ascending colon to terminate on the lateral abdominal wall. In one such case the transverse colon was drawn so sharply down as to produce a very angular flexure at the hepatic region.

According to Hall, the "structure seems to be peritoneum loosened from its close connection to the abdominal wall and colonic surface by some serous exudate after which the particular vascularization and connective tissue banding has occurred." Microscopically the membrane "seems to have its fibers split asunder by a serous infiltration, thus lifting the endothelial layer." The blood vessels are large and thin-walled. No fibrin, polymorphomuclear leukocytes or other evidence of inflammation are present. The endothelial covering in places on the surface is perfectly preserved and demonstrated beyond a doubt that we have here no new or false membrane, but simply a rarified and otherwise altered natural structure. The enlargement of the endothelial lined clefts so abundantly observed suggests a chronic lymph stasis as an associate condition. As a result of inflammation of the colon, cecum or appendix, or from the constant pulling of a dilated, prolapsed cecum, the membrane may become considerably thickened and even form organized, connective tissue.

Symptoms.—Pilcher concludes that the "membrane in all cases is a source of ill-health and suffering." He ascribes the formation of the membrane to a colitis. In that condition, there being excessive fermentation in the large gut, the latter becomes distended. The presence of a membrane binding down the cecum or colon would thus interfere with the distension and produce symptoms. We are of the opinion that the membrane is productive of symptoms only when it becomes obstructive. In fact, many membranes have been observed during laparotomies performed on patients whose signs, symptoms or disease were not all referable to the intestinal tract. In eight out of eleven cases operated upon by Cheever in which a membrane was found, the patient had none of the usual symptoms thereof. The symptoms, when present, are of the obstructive type, interfering with peristalsis and causing distension and fermentation in the cecum and ascending colon. The patients usually give a history of long-standing trouble gradually becoming of increased severity. The main symptoms are constipation, diffuse right sided abdominal pain and abdominal distension, most marked on the right side. Constipation is a fairly constant symptom, though some cases reported by Gerster complained of diarrhea. The pain at first is more of a discomfort in the region of the cecum or ascending colon, and is probably due to distension rather than inflammation. It is not associated with fever or increased pulse rate. Later it becomes more severe and occurs in definite acute attacks with intervals of comfort. The pain, though diffuse over the right side, is more marked over the cecum or hepatic flexure and only rarely referred to the epigastrium. Gastric indigestion is usually complained of, which is not influenced by change of diet, but only temporarily by a bowel evacuation. Gastric analysis does not reveal anything definite. Abdominal distension is most marked in the region of the cecum, is progressive and more noticeable at the time of an acute exacerbation. Neurasthenia from intestinal toxemia may supervene.

Physical Examination.—Inspection may show abdominal distension, with a secondary tumefaction in the right iliac region. Palpation and percussion will also reveal tympanitis, pressure over the cecum may cause considerable gurgling and afford relief to the patient by forcing gas along the large intestine. Rather characteristic is the diffuse tenderness over the right side of the abdomen without rigidity. There is often hyperesthesia over this region. Examination of the stool may show a small amount of mucus, but usually not enough to have been noticed by the patient.

The lymphatics of the cecum and ascending colon are closely associated with those of the right kidney. This fact is evidenced by the coincident cecal stasis and renal irritation or even nephritis. Pilcher reports two such cases in whom the renal condition cleared up following removal of the pericecal membrane that had caused stasis in the first part of the large intestine. He states: "Persistent pain referred subjectively to the right kidney, with some pus and blood and casts in the urine with negative X-ray findings as to stone in the kidney and positive evidence of cecal stasis, should suggest active efforts to relieve the latter as the condition of fundamental importance." The X-ray is of considerable value in the diagnosis of pericecal membranes. Before the examination the patient gets a preliminary cathasis. Some radiologists do not advise a purgative, claiming that the subsequent mobility of the colon is interfered with and thus a false interpretation of the radiograph may ensue. The patient takes a meal consisting of bismuth subcarbonate 90 grams in artificial buttermilk and water (q) S. 500 c. c. cocoa may be added to make the mixture more palatable. A bismuth enema is given in those cases in whom the filling-in effect is to be observed. Up to a comparatively recent date the radiograph was almost exclusively used. It is taken twelve hours after ingestion and at definite periods thereafter. Thus cecal ptosis, the location and extent of obstruction or angulation and ileocecal stasis are demonstrable. This ideal stasis is rather an elastic term, but if bismuth is present in the ileum after twenty-four hours the condition of stasis may be said to exist. The true value of the X-ray, however, is only appreciated by the use of the fluorescent screen, as thus we get a cinematographic picture of the intestinal motility. In the presence of a pericecal or pericolic band, if we attempt to move the cecum or colon, we will see by means of the screen that they are more fixed than normally. The filling process is characteristic, as the bismuth enters the ascending and proximinal transverse colon the latter will be pulled down toward the former, giving the double-barrel shotgun effect. These two adjacent loops cannot be separated by manipulation.

Diagnosis is based on a history of long-standing illness, increasing constipation, diffuse right-sided discomfort and distension, acute exacerbations of severe right-sided pain with increased distension, but no well marked point of tenderness and no rigidity, no pulse acceleration or temperature and a positive X-ray finding of ileocecal obstruction.

Differential Diagnosis.—Acute appendicitis is associated with temperature, pulse acceleration, localized tenderness and rigidity.

Chronic Appendicitis.—This differentiation is not always possible. There may be a history of previous attacks of fever, localized pain and tenderness, *i. e.,* typical acute appendicitis. Furthermore, in chronic appendicitis a more localized point of tenderness is elicited. Subjectively pain is often referred to the epigastrium, which only rarely occurs in membranous conditions.

Ovarian Disease.—The symptoms are more localized and bear some relation to menstruation. Vaginal examination will aid in the diagnosis.

Cholelithiasis.—There may be a history of previous gall-stone colic, presence of jaundice, pain may be referred to the shoulder. There is a very distinct tenderness over Robson's point.

Gastric and duodenal ulcer.—There is a periodic occurrence of pain in the upper abdomen, definite relation to food intake, not influenced by cathartics as in pericecal conditions, and a more definite localization.

Lane's kink.—Tenderness is usually localized at a point slightly to the left of McBurney's point. The X-ray may demonstrate the kink.

Renal colic or inflammation.—Urinalysis and X-ray findings are positive.

Prognosis.—The prognosis depends on the ability to overcome the obstructive influence of the band before secondary changes have taken place in the intestines.

LANE'S KINK AND BAND

BY HERBERT E. STEIN
Assistant Gynecologist, New York Polyclinic; Chief, Surgical Clinic, Harlem Hospital
New York

By this condition is meant an angulation of the ileum within its last four inches, causing more or less obstruction to the fecal current. This kink is usually associated with the presence of a band running from the free border of the terminal ileum along the inferior leaf of its mesentery to various points of the posterior pelvic wall. To quote from Lane: "There develops on the under surface of the mesentery of the last few inches of the small intestine a new band, which at first forms a part of the under surface of the mesentery. Later it forms a ligament distinct from the mesentery. This ligament contracts and deforms the ileum, producing a kink or obstruction of this portion of the intestine."

Etiology.—The kink and its band have been variously accounted for. Lane claims that the origin of the condition lies in a cecum which, distended from any number of cases, tends to prolapse. This descent is resisted by crystallization of lines of strain in the shape of peritoneal thickenings that later develop into independent bands. The outer bands extend from the outer aspect of the cecum upwards to the lateral abdominal wall, the lowermost often involving the appendix, with formation of a kink. Other bands run along the under or posterior surface of the mesentery of the ileum. These bands are "evolutionary and functional" (Lane). Mayo has ascribed the bands to an inflammatory condition, but admits that a considerable number showed no evidence of inflammation in the band or neighboring appendix. Martin evidently considers the band of very little importance. He believes the kink occurs within the last four inches of the ileum because of its short mesentery at this point, being only one or two inches long. Acting on both ends of this comparatively fixed loop are two forces that tend to produce the angulation. They are (1) a large gut that is excessively movable or displaced; (2) an abnormal disposition of the small gut. Thus any condition that tends to produce defective nutrition and diminish intra-abdominal fat will allow a general visceroptosis. Defective development, as described under the article on pericolic membrane, will cause prolapsed intestines.

If we now turn to embryology it will throw more light on the etiological phase. Flint states that in almost all the embryos in which he found a pericolic membrane he also found a peritoneal reflection from the posterior leaf of the mesentery of the ileum on to the parietal peritomeum near the brim of the pelvis. All these embryos except two showed a free mesentery of varying lengths. We are thus confronted with the question whether pericecal membrane and Lane's kink are not due to the same developmental factors, *i. e.,* migration, rotation and fusion of the ileocecal segment of the intestinal canal of the embryo. In fact, of the eleven adult cases of Lane's kink encountered by Flint, five also showed a cecal membrane. Three cases of Flint indicate that irregularity in embryonic fusion of the ileum or its mesentery with the posterior parietal peritoneum is a potent factor in the development of the adult Lane's kink. Connell claims that delayed migration with incomplete rotation allows the ileum to be in contact with the parietal peritoneum long enough for fusion to take place. Subsequent descent and rotation draws out this band of fusion. Excessive rotation, *i. e.,* beyond 180 deg., will produce a condition in which the ileum enters the cecum from before backwards, and is in apposition with the peritoneum at an abnormal point. Mayo claims that the lower ten inches of the ileum in its development are fixed to the lower end of the cecum by its peritoneal surface. If this fusion is not severed at birth as normally takes place, an ileal obstruction ensues. Douglas Reid has described a fetal band which he calls the genito-mesenteric fold, that runs from the mesentery of the terminal ileum into the pelvis to the genital gland. This band is found in a large percentage of fetuses after the seventh month and in its position and relation appears as the beginning of the Lane band in the adult. This genito-mesenteric band usually appears in conjunction with another fetal membrane known as the bloodless fold of Treves, which runs from the lateral parietal peritoneum across the cecum to be attached to the ileum and its mesentery and might thus cause a kinking of the ileum.

Pathology.—The ileum and its mesentery are the main sites of this pathological state. The former may show various degrees of obstruction. There may be only a rolling in of its free border owing to the upward pull of the non-yielding band on the mesentery, or the limbs of the opposing ileal loops may lie parallel or adherent to each other, in the shape of an inverted V with the apex pointing upward. Any degree between these two extremes may obtain. The result is a diminution in the caliber of the ileum, allowing of fecal stasis, fermentation and secondary changes in the structure of the ileum and proximinal intestine. The band which, according to Lane, is the main causative factor in producing the obstruction may be of varying density. It may be thickened owing to an inflammatory process of a neighboring organ. It is usually attached to the free border of the ileum within its last four inches, runs along the posterior leaf of the mesentery, where it either terminates or continues on to the peritoneum covering the psoas muscle, and then downward to the brim of the pelvis. It is interesting to note that this direction corresponds to that of the genito-mesenteric band as seen in embryo. Another band binding down the cecum is the so-called bloodless fold of Treves that crosses the cecum transversely and is attached to the mesentery of the ileum, thus forming part of a pouch for the cecum and appendix. The mesentery of the ileum is sometimes very short. The associated lesions are a varying degree of ptosis, veils covering the cecum, prolapse of the stomach or even general visceroptosis.

Symptoms.—These depend on the extent of obstruction produced by the kink or angulation. There may be no discomfort or acute and fatal obstruction. The latter, however, is usually of a chronic nature, characterized by distress in the lower right abdomen near the umbilicus, constipation of increasing severity or periodic attacks of colic in the appendicular region. These attacks of pain usually bear definite relation to the time of taking food, are aggravated by excessive moving about, by fatigue or constipation, but relieved by an enema or by the reclining position. Later on the pain becomes more or less continuous. These attacks are often associated with anorexia and gaseous distension. Backache due to a pull on the mesentery is sometimes complained of. Symptoms of toxemia and weakness supervene. Some of the symptoms of autointoxication as enumerated by Lane are: Loss of fat, wasting of the muscles, skin pigmentation and offensive perspiration, subnormal temperature; apathy, stupidity or even melancholia, neuritis, headache, neurasthenia; pains in muscles and joints, atrophy of thyroid, shortness of breath, symptoms of cardiorenal, liver and pancreatic disease.

Physical examination may show a general condition of faulty development, abdominal distension, cecal dilatation and in many cases a characteristic point of tenderness one inch below and to the right of the umbilicus. X-ray examination will demonstrate ileal stasis and abnormal fixation as shown by its manipulation when viewed through the screen.

Diagnosis.—Lane's kink is characterized by a history of chronicity, periodic attacks of partial obstruction, associated with colicy pains and tenderness just to the right of and below the umbilicus. X-ray examination will reveal the obstruction.

Differential Diagnosis.—Acute appendicitis is associated with temperature and pulse acceleration, the tenderness is at McBurney's point and not so near the umbilicus, and the abnormal rigidity is more marked.

Chronic Appendicitis.—As this condition is often associated with Lane's kink the differentiation is frequently impossible. The X-ray examination and the dissimilarity in the points of tenderness may be of value.

Tubo-ovarian Disease.—The history, relation to the menstrual period and vaginal examination will usually clear up the diagnosis.

Prognosis.—As the band is not inflammatory it will not tend to reform after being severed; but cure of the concomitant conditions will depend upon the subsequent management of the case.

Treatment.—Through a right rectus incision the band is severed, thus allowing the ileum to unroll and straighten out. It is advisable to cover over the raw surfaces with peritoneum. To overcome the enteroptosis, a fattening diet and abdominal massage are prescribed and an abdominal support worn. Daily assumption of the Trendelenburg posture is valuable.

THE PROGNOSIS OF PULMONARY EDEMA

By THOMAS F. REILLY
Professor of Applied Therapeutics, Medical Department, Fordham University
New York

If one takes the view point of the older pathologists, particularly those of the Vienna school, that patients do not die of pulmonary edema but get pulmonary edema because they are dying, there can be but one prognosis in any one case. Most clinicians of the present day will disagree with this dictum, because every one has seen some cases of pulmonary edema recover and apparently get well and remain so, at least for months. In addition, one sees quite a fair proportion of other cases recover temporarily, only to pass away within a few days.

Probably the reason for these divergent facts is that it is often a matter of definition as to what constitutes pulmonary edema. If one postulates that every pulmonary edema must always have generalized liquid râles, dyspnea, and frothy sputa, then recovery will not be so common; on the other hand, there are many cases, particularly in patients suffering from early cardiac decompensation, who present evidences of an edema of the lungs, but there are not enough signs to completely fill out the picture as just outlined. There is neither much dyspnea nor is there frothy expectoration present, but technically there is some edema of the lungs. They are classed as congestion by some authors, but, as in any severe pulmonary congestion, there is always more or less edema present, the tendency is to agree with McKenzie and call such edemas pulmonic. These cases almost always bear a good prognosis provided we can cause the cardiac mechanism to overcome the decompensation. Fortunately, this is almost always the case, at least in the early attacks. Later on, with frequently occurring congestion, the edema may slowly spread; but even here it is not until there is kidney involvement with general anasarca that pulmonary edema is a terminal event. Even in these latter cases there is a fair modicum of hope under proper treatment, so that in general when one is called for the first time to see a patient suffering with pulmonary edema, either as the result of an acute dilatation or of an acute exacerbation of a chronic cardiac process, provided one finds a valvular lesion present, he is warranted in giving a fairly hopeful prognosis. At any rate, it is not necessarily terminal. Of course, occasionally a patient dies in such an attack, but the number of recoveries occurring among patients who are brought into an active ambulance service and who are suffering in this manner makes one feel that edema of the lungs due to a mitral valvular lesion is not so hopeless as it looks.

Of course, the matter of treatment has much to do with the prognosis. Patients who have *not* been under treatment, and live under unfavorable circumstances, do react more positively when brought under institutional care than those who have been under long and careful treatment. In the one instance, the wells of treatment have scarcely been tapped and a quick response may be expected to proper medication; in the other, every means has been pretty thoroughly applied and there is less spring or reaction to treatment.

Pulmonary edema is a frequent cause of death or terminal event in the course of apoplectic strokes, whatever the cause of the stroke may be. In my experience, those due to cerebral syphilis are less likely to be followed by pulmonary edema than the others. When the edema occurs within the first few hours, recovery is possible, but not likely. Sometimes such an edema does clear up for a day or two, and one begins to build false hopes, but in most instances the patient gradually sinks and dies.

When edema comes on after the second day, it is always fatal. Of course, when this edema is due to an accompanying cardiac lesion, there may be a ray of hope. During and after an uremic convulsion, one frequently finds all of the evidences of pulmonary edema. Such cases are quite frequently recovered from, although it must be admitted that the vast majority perish. If the patient quickly regains consciousness, say within two or three hours, it has seemed to us that the outlook is in most cases favorable. Occasionally when coma or partial consciousness has lasted a day, a patient will recover from the edema, but as a rule, even this one will succumb after a few days.

Absence of very high tension seems favorable to recovery. If a moderately large amount of urine is excreted, the outlook is more promising. Prompt and active treatment has much to do with the outlook in these cases, and in no class of pulmonary edema cases does one more often experience the chagrin of seeing a patient alive and apparently well who had been condemned to certain death because of the presence of severe pulmonary edema occurring during an uremic convulsion.

Of the cases occurring in the course of toxic states, those associated with pneumonia comprise by far the greater percentage, when the edema is local, *i. e.,* when it only affects the rest of the affected lobe,—a collateral edema; it increases the gravity immensely and recovery is not the rule; but when it involves the rest of the lung on the other side it is invariably fatal.

Occasionally one meets with a pulmonary edema that develops early in the course of a pneumonia. This is especially apt to happen in an old patient. With active stimulation, this often will clear up temporarily and the patient may be fairly comfortable for a few days, but he generally succumbs later. When generalized pulmonary edema develops late in a pneumonia, there is only one ending.

In general, these remarks obtain in the case of other toxemias—*i. e.,* in the early stages, temporary recovery is possible, complete recovery is exceedingly rare; but when it occurs late in the disease there is no hope.

The same prognosis applies to those developing in the course of the cachexias, such as Hodgkin's disease, carcinoma, pernicious anemia, etc.

Patients with enlarged thymus do have attacks of pulmonary edema, but they generally recover from the early attacks only to perish later of some intercurrent trouble.

One occasionally meets with a pulmonary edema in a middle-aged patient as a result of alimentary toxemia. These cases usually have some indefinite indications of an underlying renal process, such as a slight or transient albuminuria, slight rise in pressure, but nothing definite. With the exception of frothy sputa, the picture is complete and death seems imminent, yet under treatment recovery is the rule, and the attack may not repeat.

In the acute, recurring, or paroxysmal pulmonary edema, associated with cardiovascular disease, death is uncommon in the first attack. In one case under my own observation the patient was said to have had the attacks for forty years prior to the attack in which he died. The average duration of life in the cases seen by me have been about two years after the first attack. The patients do not generally die during the attack itself. There is a class of these cases where death occurs always within the first five or ten minutes, and for which nothing can avail. These closely simulate pulmonary embolism, occur in old people and are characterized by a vast amount of frothy expectoration, which continues to well out after death. For the most part, however, they commonly succumb to the symptoms of gradual loss of compensation. When edema of the legs develops, the attacks, even though they recur, are much milder than previously. As far as the attack itself is concerned, cases that are going to get well do not progressively grow worse after the first ten or fifteen minutes. When the pulse tension falls early during the attack, the outlook is very serious. When it falls later, with coincident improvement in the respiration and other symptoms, it is a good sign. According to Dieulafoy, absence of expectoration is a bad sign, yet if it was found present early in the attack and then disappears, it is a good omen.

This has not been in accord with my own observation, as I have been unable to see that the cases without expectoration were any worse than the others. In fact, they seemed to be more easily managed.

French observers believe that when a number of mild attacks precede the grave attack on the same day, it is more often fatal than otherwise.

When one hears a clanging second sound at the right of the sternum, the outlook is good.

In general, the more intense the vascular phenomena the worse the prognosis. In severe cases, the fall of blood pressure is more rapid than the rise, thus showing cardiac failure. In these cases, the apex beat grows weaker, the high tension pulse becomes weaker, more rapid, and finally thready; recovery seldom occurs in such a case.

The average duration of untreated attacks is from three to four hours. Of course, there is some prostration over a longer period. Under active treatment the paroxysm is over in an hour or two and the patient is fairly comfortable, and the physician may leave him in about one half hour.

There are some recorded cases where death has occurred within fifteen minutes; and in others, where the duration of the attack has been as long as twenty-four hours. When an attack follows another in rapid succession for an interval of three days or more, recovery seldom occurs.

When pulmonary edema develops as a result of the use of pilocarpine in uremia, recovery is rare. It must be admitted that in such cases pulmonary edema might have developed anyhow, but there is sufficient evidence to make it certain that pilocarpine can in certain cases produce pulmonary edema.

In the cases associated with angioneurotic edema the prognosis is accounted to be good.

When pulmonary edema follows thoracic paracentesis the outlook is almost always favorable. In such cases, only one lung is involved, and while the amount of frothy material expectorated may be considerable, yet after the first few minutes these cases are not alarming; death, while it does occur, is very uncommon.

When pulmonary edema follows head injuries, recovery is a rarity. Occurring in pregnancy and labor, it constitutes one of the modes of death in cardiacs, particularly those suffering from mitral stenosis. When it does happen, it is usually during or shortly after labor itself.

Statistically: In one series or five thousand labors, there were eighty cases of heart disease, and among these were eight deaths. Of these, two died of pulmonary edema.

In another set of statistics there were three deaths from pulmonary edema in thirty thousand pregnancies, so that, statistically, it is not as common as one might think. It is often confounded with pulmonary embolism.

When pulmonary edema follows the employment of large saline infusion it is almost always fatal. Here, again, the question of cause and effect always bobs up inasmuch as the disease for which the infusion is done may be the cause of the edema.

Following muscarine poisoning, at least experimentally, it is almost always recovered from by the prompt administration of the antidote, atropin. Occurring after ether anesthesia, the prognosis is fairly good.

From a review of the literature it appears that about fifty per cent. recover. Here again the question of what shall be called pulmonary edema is an open one. Such a large proportion of cases following ether anesthesia have large and small liquid râles scattered over the whole chest, have slight increase in pulse, and some dyspnea, that it may be a nice question to decide if pulmonary edema is present or not. Inasmuch as these symptoms pass off on return of consciousness, it is hardly fair to class them as edema. Those who specialize in anesthesia consider it a very rare complication.

One not infrequently finds the sign of pulmonary edema in children during an infantile convulsion. Here the outlook is quite good, the edema disappearing within an hour or two after the return to consciousness. In fact, all edemas associated with convulsions are improved and tend to disappear on the return to consciousness.

Pulmonary edema in children, excluding those associated with convulsions and those rare cases associated with enlarged thymus or obstructive disease, is associated with the infections, particularly pneumonia. In these cases the onset is usually quite sudden and death occurs usually within a few hours at most. I have never seen recovery occur in a well developed case.

Finally, in conclusion, I believe that he who possesses most therapeutic hopefulness in his make-up will have most success in his cases of pulmonary edema. True, this percentage will not be numerically a large one, yet there is a substantial percentage of recoveries.

Excepting in diseases for which we have specific remedies, I feel that therapeutic nihilism spells death here more than almost anywhere else in medicine. Even though the patient recover temporarily and death is but postponed for a day or two, yet there is ample justification for our professional services in such a case. Certainly, the sting of death itself, in the shape of mental anguish, is taken out of the minds of the relatives and friends who witness the recovery from pulmonary edema, even though it be but for a short time. They do not feel it so badly as though death occurred in the first instance.

CHRONIC BRONCHITIS, EMPHYSEMA AND ASTHMA

By J. EPSTEIN
New York

Chronic bronchitis, emphysema and asthma form a group of diseases which causes a great deal of confusion in the diagnosis and in the differential diagnosis. Errors in the diagnosis of this bronchopulmonary triad are due to the fact that this entire group of diseases may be secondary to some other underlying disease, and the proper diagnosis is that of the primary disease with the bronchopulmonary affection as a complication or a sequela. Chronic bronchitis, emphysema or asthma may be secondary to diseases of the nasopharynx, chronic pulmonary infections, cardiovascular diseases, aneurism of the thoracic aorta, mediastinal growth, and constitutional diseases such as syphilis, gout, nephritis, alcoholism or obesity. It is essential for scientific as well as for practical therapeutic reasons to decide in every case whether the bronchopulmonary affection is a primary disease or is secondary to some underlying disease which is obscured by the more prominent symptoms and signs of a chronic bronchitis, emphysema or asthma. A detailed history and careful physical examination especially of the lungs and the cardiovascular systems with the aid of the laboratory, will lead to a proper diagnosis and to successful treatment.

The differential diagnosis between chronic bronchitis, emphysema and asthma is frequently erroneously made because this entire group of diseases or any two of them may be present at the same time or any one of them may complicate the other or be a sequel to it. Patients with chronic bronchitis, emphysema or asthma have many points in common in their family history, past history, their complaints of cough and dyspnea, and in their symptoms and physcial signs, yet they are separate and distinct diseases and for diagnostic as well as for therapeutic reasons it is important to make an exact diagnosis in each case and when two or all the three affections are present to diagnose the first or primary disease of this bronchopulmonary triad in order that treatment may be applied to the source of the trouble.

Chronic bronchitis is a disease of the bronchi, emphysema is a disease of the pulmonary tissue, asthma is a disease of the neurobronchial system.

In chronic bronchitis, the bronchi as well as the peribronchial tissues are affected, and whatever the underlying pathologic condition may be, there is a bacterial infection of the mucous membrane with some of the common organisms as the staphylococcus, streptococcus, pneumococcus, influenza bacillus, bacillus catarrhalis and certain saprophites. The patient complains of cough and dyspnea worse in cold weather and after more or less exertion. The physical findings in an uncomplicated case of chronic bronchitis are not many and the diagnosis is not difficult. On inspection there is usually some dyspnea which may be inspiratory or expiratory or both, depending probably on the location and the size of the bronchi chiefly affected; the lower the disease is in the bronchial tree the greater is the expiratory difficulty, the higher it is the greater the inspiratory difficulty. On palpation and percussion there may be nothing abnormal, while on auscultation there are usually some dry and moist râles. Bronchiectasis, atelectasis, or chronic pulmonary fibrosis may complicate chronic bronchitis and make the diagnosis difficult.

Emphysema is a disease of the pulmonary tissue, specially the elastic fibers, and is in the majority of cases secondary to chronic bronchitis or asthma. The patient is cyanotic and looks emphysematous, he complains of cough and great dyspnea which affects chiefly the expiratory phase of respiration. In emphysema the lungs are too large for the chest and the chest appears too big for the patient. On physical examination one finds in addition to a chronic bronchitis or asthma a hyperresonant chest with the borders of the lungs extending beyond their normal limits, diminished cardiac and hepatic dulness, diminished breath sounds with prolonged expiration and a variety of râles.

Asthma is mainly a disease of the neurobronchial apparatus, whatever the underlying cause may be which brings about the periodic attacks of the bronchial spasms. The patient complains of severe dyspnea, both phases of respiration being affected, specially the expiratory phase. There is a condition of air hunger in spite of the fact that the lungs are overdistended with air. The diagnosis can be made on inspection. Palpation, percussion and auscultation will elicit rhonchial fremitus, hyperresonance and a conglomeration of dry and moist râles of different degrees and musical qualities. In a difficult case the increased number of eosinophiles in the blood and the Curschmann's spirals with the Charcot-Leyden crystals in the sputum will help in the diagnosis. It is essential to make a clear distinction between asthma and dyspnea. While asthma is dyspnea, dyspnea is not asthma. Asthma is caused by bronchial spasms and is periodic in its attacks, dyspnea may be caused by many diseases, specially by cardiac and renal affections and is more or less constant. Attacks of dyspnea in youth and middle age is likely to be asthma, while after middle age it is mostly of cardiovascular origin and is relieved not by antispasmodic drugs but by strychnin, digitalis or strophantus.

The lungs and the heart are intimately associated anatomically and functionally and are the most interdependent organs of the body. In chronic diseases of the bronchopulmonary system the heart suffers in its effort to overcome congestion or obstruction in the bronchopulmonary circulation, and in diseases of the heart the bronchopulmonary system suffers from an imperfect or congested circulation, the bronchi being mainly affected because the blood from the bronchi returns through the bronchial veins into the right side of the heart and through the pulmonary veins into the left side of the heart. Affections of either side of the heart causes stasis in the bronchi. It is therefore essential that in diseases of either the respiratory or the cardiovascular system, that both systems receive careful examination and treatment.

The following few brief cases will serve to illustrate the frequent errors in the diagnosis of chronic bronchitis, emphysema and asthma.

Case 1. A man, 35 years old, single, laborer, consulted me because of cough and dyspnea from which he suffered for about six months before he came under my observation. There was nothing of importance in his family and past history; he denied venereal diseases. He was treated for chronic bronchitis and asthma. On examination I found slight dulness in the base of the left lung, diminished breath sounds, with some dry and moist râles all over the left lung. The right lung gave no evidence of any disease. To account for the unilateral involvement of the entire left lung further examination revealed a fairly large aneurism of the thoracic aorta which probably caused pressure on the left bronchus and gave rise to the cough, dyspnea and the physical signs. The rest of the examination was negative. A positive Wassermann test of the blood and an X-ray of the chest confirmed the diagnosis of aneurism.

Case 2. A woman, 50 years old, came to me with a ready-made diagnosis of asthma and emphysema for which she was previously treated. There was nothing of importance in her family history. Had typhoid fever eight years before. She complained of slight cough and considerable dyspnea. Could not sleep at night but had to sit up near an open window to get in some air. On examination there was nothing abnormal in her lungs, but her heart gave every evidence of myocardial insufficiency. Abdomen negative, urine negative, and the blood pressure was normal. A proper diet, regular bowel movements and 10 drops of tincture of digitalis twice a day caused a great improvement in her condition.

Case 3. A woman, 32 years old, had pneumonia two years before and was coughing since. Had lost some weight and did not feel well. A diagnosis of chronic bronchitis was made; she was treated for this disease for some time. On examination I found dulness in the right apex with numerous râles all over both lungs. The heart and the abdomen were negative. I made a diagnosis of pulmonary tuberculosis which was substantiated by a positive sputum.

Case 4. A boy, 4 years old, was brought to me for the treatment of cough. The family history was negative. He was never sick before, except that he has been coughing for the last year. He felt better during the summer months, but was worse in cold weather and coughed a great deal at night. Chronic bronchitis was the diagnosis for which he was treated. Examination revealed a chronic nasopharyngitis and tonsillitis. The lungs showed nothing abnormal except that inspiration was rather prolonged and was of a peculiar snoring quality, which gave one the impression of râles and together with the cough lead one to a diagnosis of bronchitis. A mistaken diagnosis of bronchitis is frequently made in children with chronic nasopharyngitis and tonsillitis because of the chronic cough and the rough sonorous breathing. This kind of breathing is probably due to the fact that the air during inspiration meets with an uneven and partly obstructed surface in the nasopharynx which imparts to it a stertorous quality and is transmitted along the bronchial tree like râles. It can be easily differentiated from bronchitis by the fact that this râles-like sonorous breathing is heard alike all over the chest and is everywhere of the same character and quality, showing that it must come from a single source in the upper respiratory tract. Treatment applied to the hypertrophied tonsils and nasopharynx will cure the supposed chronic bronchitis.

DIAGNOSTIC ERRORS IN SENILE CASES

By I. L. NASCHER
New York

My purpose in presenting this article is to arouse an interest in geriatrics, the branch of medicine dealing with the aged and their ailments. I wish to show how, owing to our lack of knowledge of the senile organism in health and in disease, our failure to differentiate between the normal senile degenerations and disease conditions, and our ignorance of the interrelation between the processes of senile involution and pathological processes, we have a wrong conception of disease in old age, we make errors in diagnosis and consequently in treatment of our senile cases.

A pathological process or condition is a deviation from the process or condition normal at a certain period of life. The infant's weakness, its inability to talk, to walk, to reason, are physiological at that period of life; occurring at a later period they would be pathological. We look upon childhood as a physiological entity, apart from maturity, and treat the child as a child and not as an adult with undeveloped organs and tissues. If we were to look upon senility as we look upon childhood, as a physiological entity, and not as a pathological state of maturity, we would get a new conception of disease in old age. We would then realize that disease in old age is a pathological process in a normally degenerating organ or tissue and not a pathological process in an organ or tissue as it exists in maturity, complicated by degenerations. We would realize that the senile degenerations are not diseases and we would recognize the folly of attempting to restore degenerated or diseased senile tissue to the condition normal in maturity.

The principal sources of error in diagnosis in senile cases are (1) considering the senile degenerations as diseases; (2) mistaking the senile degenerations for diseases which they resemble; (3) overlooking symptoms of disease which are masked by the manifestations of senility; (4) unreliable history, ill-defined symptoms and atypical diseases; (5) misinterpretation of signs, symptoms and symptom-complexes.

Under the first heading, considering senile degenerations as diseases, the most prominent example is senile arteriosclerosis. In a previous article on "A New Conception of Arteriosclerosis," I pointed out eight distinct forms or types of arterial degeneration associated with hardening of the vessel. Among these is the mechanical form or type which includes senile arteriosclerosis. In this form the unceasing contraction and relaxation of the muscular fibers of the arterial walls, over one hundred thousand times a day, causes a loss of tonicity and waste of the muscular fibers. In early life this waste is immediately repaired in the ordinary process of metabolism. Later, repair is slower and in advanced life repair is incomplete, the waste being partly replaced by fibrous connective tissue. In advanced life the loss of tonicity of the muscular fibers which are not immediately repaired, permits a dilatation of the vessel with diminished resilience, and the repair by fibrous tissue causes a hardening of the walls. This fibrosis is a normal physiological senile degeneration. There are also other factors involved in the production of senile arteriosclerosis, but atonicity and waste of the muscular fibers and succeeding fibrosis are the fundamental factors. Failure to differentiate between this form and the irritative or inflammatory form, the usual pathological form which begins as an endarteritis, is responsible for the many futile attempts to cure senile arteriosclerosis and restore the hardened vessels to the condition normal in maturity.

Medical literature abounds with treatments of arteriosclerosis. The very fact that some forms of treatment are successful in some classes of cases, other forms of treatment are successful in other classes of cases, and no form of treatment is of the slightest benefit in senile arteriosclerosis, except as a momentary palliative, indicates that there are different forms of the disease and the senile condition is not susceptible to cure in the sense of restoration to the condition normal in earlier life. It is almost always possible to reduce the high blood pressure which is one of the symptoms of this condition, but the relief of a symptom does not restore degenerated tissue. In differentiating between senile arteriosclerosis and the pathological forms we must take into consideration the etiology, symptoms, signs and results of treatment. Senile arteriosclerosis is always primary, is diffused, comes on late, progresses slowly and is accompanied by other signs of senile degeneration, as phlebosclerosis and venosity, hardening of the xiphoid cartilage, contracted kidney and other manifestations of senility. The blood pressure follows the formula, age plus 100 in millimeters for the systolic pressure at the patient's age. The arteries are hard and tortuous but not beady. Iodides will temporarily reduce the blood pressure, but the physiological action of iodism, bronchial and nasal catarrh, gastric disturbances, rashes, soon appear and the drug must be discontinued, when the blood pressure will almost immediately rise to the normal pressure at the age of the patient. The irritative or inflammatory form of arteriosclerosis is always secondary to a toxemia, either nephritis, diabetes, gout, syphilis or other infectious disease or other endogenous or exogenous toxin. It proceeds rapidly, is frequently localized, there is no marked phlebosclerosis, venosity or other manifestation of senility, the blood pressure rises rapidly, usually above the upper limit of the normal at the age of the patient, it falls rapidly under treatment or as the disease improves unless the degeneration of the vessels is far advanced. The arteries are hard, generally beady, but they do not become as tortuous as in senile arteriosclerosis. There are structural differences between the various forms which can be differentiated by pathologists.

Various visceroptoses which are normal in the aged and due to flaccid diaphragm and flaccid and wasted abdominal walls, are usually treated as diseases. Gastroptosis and dilatation of the stomach is the normal condition of the senile stomach. The liver sinks, its lower border being sometimes on a level with or even below the umbilicus. Enteroptosis is usual and is generally a contributing cause of senile constipation, the principal causes being diminished peristalsis through atony and waste of the muscular fibers, and diminished intestinal secretions. These ptoses can be relieved by mechanical supports, but they cannot be cured, although they may be altered by surgical means. Senile emphysema, with its dyspnea, wheezing respiration, and general symptoms of impaired aeration, is sometimes treated as a disease. Its progress may be retarded by the use of braces, corsets, cane, or other mechanical appliances, and living in a dense, dust-free atmosphere, but nothing will restore the wasted alveolar septa. Let me mention here a pathognomonic symptom of this condition. After the patient has been lying upon his back for some time, if he will sit up and take a deep breath, fine crackling râles will be heard close under the skin during the deep inspiration and perhaps during the next inspiration or two, but not later. The râles are due to the opening of the air vesicles which had been compressed while the patient had been on his back, and open up during the first few deep inspirations.

Brown atrophy, while very rare, is a true senile degeneration not amenable to treatment. Cardiac hypertrophy, the usual condition of the heart in old age, is a compensatory condition made necessary to overcome the hardening and lessened elasticity of the arteries, the increased peripheral resistance and probably the increased density and viscosity of the blood.

Intestinal degeneration, in which the peristalsis is diminished through atony and waste of the muscular fibers and diminished intestinal secretions, and evidenced by constipation, is generally treated as a disease. If the individual has a stool without the use of cathartics every second or third day, nothing should be done to produce a daily stool, as this is the normal condition in that person. There may, however, be a stool every day or every second day and yet the stools are delayed, undigested food particles requiring two or three days to pass through the intestines. This requires treatment.

Under the second heading, mistaking senile degenerations for diseases which they resemble, senile arthrosclerosis is a prominent example. This is a hardening of the joints due to hardening of the cartilages, tendons and ligaments and roughening of the articular surfaces. This condition, which is most pronounced in the ankle joint, is almost always diagnosed and treated as chronic arthritis. In the pathological condition the joint is usually swollen, tender on pressure, becomes stiff during rest, painful when first moved, but the pains lessen upon motion or limbering up, as the patient calls it. In the senile condition the joint is not swollen or tender, rest gives relief, there is no pain when the joint is first moved after rest, but prolonged motion causes pain and increasing stiffness. After prolonged standing or walking there may be a hypostatic edema, but this is not the same as the swelling of chronic arthritis.

The normal senile kidney is frequently mistaken for the kidney of chronic interstitial nephritis. I might refer here to the frequent reports of the pathologist's findings of chronic interstitial nephritis in senile cases which gave no history of the disease during life, and the frequent diagnosis of chronic interstitial nephritis in senile cases, which was not corroborated upon autopsy. The normal senile contracted kidney shows upon autopsy a uniform fibrosis throughout the organ; the pathological kidney shows scattered areas of hyperplasia besides tubal degeneration. Clinically the normal senile kidneys have a daily output of 1,000 to 1,200 c. c. in males and from 900 to 1,000 c. c. in females; specific gravity never below 1,010, usually between 1,015 and 1,025. The output in chronic interstitial nephritis is from 1,000 to 1,500 c. c., specific gravity, 1,002 to 1,012, rarely higher. A trace of albumin may be found in both conditions, but casts and blood elements are never found in the normal senile condition. Many of the symptoms usually connected with interstitial nephritis may be found in senile cases, but if each symptom is traced to its source it will be found that most if not all are due to the normal senile degenerations and not connected with the kidneys.

Senile cardiac myofibrosis is often diagnosed as chronic myocarditis. The senile condition is always primary, its antecedents being cardiac hypertrophy and coronary arteriosclerosis. Chronic myocarditis is always secondary to an acute myocarditis generally following infection. In both conditions the heart becomes irregular under excessive activity and regular again upon rest, but in myocarditis the heart becomes stronger during rest, while in the senile condition rest will quiet the heart but it will not strengthen it. The response to digitalis is rapid and usually lasting in myocarditis, slow and ephemeral in senile myofibrosis. Upon autopsy the diseased heart shows pigment infiltration which is absent in the normal senile heart.

The senile liver, which is smaller and firmer than the liver in maturity, is sometimes mistaken for the late stage of cirrhosis of the liver, and it may be mistaken for waxy degeneration or cancer, the diagnosis being based upon the physical signs. In the absence of a history pointing to one of these diseases such an error should not be made if the physician will remember the senile changes in the liver. Occasionally a diagnosis of pleurisy will be made upon the discovery of pleuritic friction sounds, especially if accompanied by the physical signs of senile (atrophic) bronchitis, and the short, distressing cough accompanying the latter.

The pleuritic friction sounds in the aged are generally due to absence of the pleuritic exudate and the drying of the pleura.

Senile tremor, a late manifestation of senile involution, is sometimes mistaken for paralysis agitans. It comes on much later than the pathological condition and there are absent the characteristic attitude, gait, position of the hands, immobile face, forward pitch, the cramps, and muscle stiffness.

The manifestations of senility sometimes mask the symptoms of disease. Diseases which in maturity give clear, well marked symptoms, may, in the aged, produce symptoms so mild and ill-defined as to be overlooked and the physician, finding only the pronounced symptoms of senile degenerations, will treat these conditions while the disease is neglected. As the senile degenerations are slowly progressive, the aged person becomes accustomed to the discomforts connected with them and does not complain about them. When an aged person complains of not feeling well, or he exhibits restlessness or irritability not present before, or he mutters in his sleep and has never before done so, there is some disease present though it gives no clear symptoms. It is necessary in such cases to eliminate as far as we can in our determination of the diagnosis those symptoms which have been present for years, slowly progressing and not markedly increased during the disease, as these are the symptoms of senile degenerations. Only such symptoms as have not appeared before the malaise or other indication of disease, and such symptoms of senile degeneration as have become rapidly more pronounced, should be considered in forming the diagnosis. While we may be able to disregard the symptoms of senility, we are not always able to eliminate the signs. We cannot eliminate, for example, the hyperresonant percussion note of senile emphysema which might mask the percussion dulness of pneumonia. A patient complained that he felt sick all over but had no pain anywhere. Examination revealed a distended abdomen and the patient declared that his abdomen was always distended when he was constipated and he had no stool for two days. A cathartic had no effect except to produce cramps. Collapse followed and autopsy revealed an incarcerated hernia. The only suggestion that can be made in cases like this is to disregard the symptoms arising from the normal senile degenerations and look for some pathological condition.

A frequent source of error in diagnosis in senile cases is the unreliable history as given by the patient. Owing to weakened memory the aged patient will forget early ailments and may deny early diseases in spite of the presence of pit marks, scars of operations and scarifiers, etc. Present symptoms may be exaggerated or symptoms may be fabricated to obtain sympathy, or, on the other hand, symptoms may be suppressed and denied if fearful of a painful examination or operation or in a spirit of perverseness or stoicism. The patient will declare that he is unable to sleep, yet close questioning will show that he takes frequent naps during the day and he thus gets from three to four hours' sleep in short stretches, then retiring at eight or nine o'clock he awakes in the early morning hours and cannot sleep longer. He has had eight or nine hours sleep in the 24 hours, all he requires. He may declare that he has not eaten a bite all day and has no appetite, yet we may find that he had taken frequent nibbles of food, the whole amount being as much as he would have taken in two or three full meals. It is often necessary to obtain the history of the case from the family, but such information should never be sought in the presence of the patient.

The uncertainty of the subjective symptoms as obtained from the patient, and ill-defined objective symptoms, cause frequent errors in diagnosis. The difficulty at arriving at a correct diagnosis is increased by the modification of diseases in old age.

The infectious diseases are rare and always grave, yet the aged may be carriers of infection. Typical temperature curves, as in typhoid fever and malaria, are seldom found in the aged.

Pain is frequently absent in diseases that are usually painful in earlier life, as in pneumonia, pleurisy, peritonitis, appendicitis, which latter, by the way, is very rare in the aged. While aged persons will complain of general discomfort and distress, of headache, bellyache, backache, pain all over, they seldom localize the pain. They will frequently deny the presence of pain upon pressure, although their facial expression will indicate a spot painful when pressed upon. Indeed, the facial expression will often give more reliable information than their statements. Constipation is the usual condition of the bowels and the aged become so accustomed to this condition that they often do not know when they had a stool last. Nor do they know much more about the passing of urine. We can get a fair indication of the condition of the bladder from the way the urine is passed. As the tonicity of the bladder diminishes the force with which the urine is voided is lessened and in very old age it may drop from the meatus. If there is a delay in starting the flow and it then comes out in force there is some obstruction, either stone, hypertrophied prostate, or old stricture. Reflex vomiting is rare in the aged and only some intense irritation of the stomach wall will cause vomiting. Nausea may accompany any condition which makes the old person feel ill. The reflexes are uncertain symptoms, as they are sometimes exaggerated, sometimes diminished in normal senile individuals.

Perhaps the most frequent source of error in diagnosis in senile cases is misinterpretation of signs, symptoms and symptom-complexes. There are many diseases which give a fairly pathognomonic symptom-complex and which usually can be diagnosed from the clinical history alone. In the aged every symptom must be traced to its source before we can determine its relation to the suspected disease. Many of the symptoms usually associated with chronic interstitial nephritis and diabetes occur normally in the aged as manifestations of senility. While the positive diagnosis in many such diseases would depend upon the laboratory findings, there are cases where X-ray and other laboratory facilities are not available and the physician must depend upon the clinical history alone. Let me present a typical case. An aged woman applies for treatment for an intense vulvar pruritus. Questioning brings out this clinical history. She is losing weight and strength; appetite good; drinks six or seven cups of tea a day, because her throat always feels dry; passes urine half a dozen times a day; constipated. The vulva shows scratch excoriations and there is intertrigo in the groins and under the breasts. The tongue is red, skin dry, face sallow. Here is a perfect picture of diabetes requiring only the laboratory examination to determine the percentage of sugar. A tentative diagnosis of diabetes is made until the laboratory report is received. The report is negative. The progressive emaciation and weakness and the dry skin are normal in old age. Pruritus is frequently found in the aged and seems to be due to some degenerative process in the nerve terminals in the degenerated skin. The frequent urination is due to atony of the sphincter, a frequent trouble with old women. The dryness of the throat, which occurs frequently in the aged, was in this case aggravated by the excessive use of tea which she drank to relieve this dryness. She was otherwise careful in her diet but had accustomed herself to periodical cathartics.

I will close this paper with a review of some signs and symptoms of disease in the aged which are frequently misinterpreted.

Temperature. The rectal temperature in the aged is usually a degree less than in maturity and the mouth temperature may be from one and a half to two degrees less than the rectal temperature. A mouth temperature of 99 deg. F. may therefore be three and a half degrees above normal.

Pulse. A hard, irregular pulse may occur in arteriosclerosis with myofibrosis, beats being lost in transmission.

Blood pressure. A blood pressure corresponding with the formula, age plus 100 for the age, indicates that the arterial degeneration and the cardiac compensatory hypertrophy are progressing normally.

Pain. Reference has been made to the uncertainty of this symptom in diagnosis. Anesthesia may be due to mental impairment.

Eyes. The pupils are generally contracted and if the aged person with presbyopia tries to look at a near object without his glasses he stares and has the appearance of being in pain.

Face. The face is usually sallow and there may be areas of passive congestion over the malars, resembling the flushes of pneumonia.

Body. The skin may look muddy, as the aged dread the bath, especially if obese. They find it difficult to get in and out of the tub.

Heart impulse. This may be strong notwithstanding a myofibrosis if the intercostal muscles are wasted. The heart sounds, and neither the pulse nor the impulse, gives the character of the heart.

Vertigo. If due to arteriosclerosis it appears at irregular intervals and is not affected by light, sound, reading or position. Many cases of vertigo in the aged are due to gastric disturbance.

Dribbling of stool and urine. If both occur it is due to the weakening of the subconscious control over the sphincters and indicates mental impairment, though the reasoning faculties appear sound.

Spleen. This organ is greatly atrophied in the aged and it may not be felt or determined by percussion in diseases usually accompanied by splenic hypertrophy.

Gastric crisis. Old persons sometimes give a history of occasional intense pain in the stomach or to the right of that organ, resembling gall-stone colic. In most cases this is due to abdominal arteriosclerosis. It occurs at a much later age than gall-stone colic and is not accompanied by jaundice or clay-colored stools.

I have mentioned here but a few of the many symptoms occurring in disease in the aged which are frequently misinterpreted and thus lead to errors in diagnosis in senile diseases. I hope I have been able to show how we, owing to our ignorance of geriatrics, make mistakes in the diagnosis and treatment of our senile cases. I hope I have been able to arouse in you an interest in the aged and their ailments; hope that as a result of such interest we may make a closer study of this neglected class, who certainly deserve all the care and attention that we can give them to make their closing years comfortable and happy.

A SYMPTOM IN EXTRA-UTERINE PREGNANCY

By HENRY J. LEHNHOFF
Lincoln, Nebraska

The symptomatology and physical findings in extra-uterine pregnancy have been frequently reviewed. It is not the object of this brief paper to take up that general subject.

Six years ago I was called to a case presenting an acute abdominal condition. Two physicians who preceded me could not agree on a diagnosis. I had the advantage of seeing the case two or three days later when a diagnosis of extra-uterine pregnancy was easily made. The patient was operated upon. A large hemorrhage had occurred.

About six weeks later I saw another woman whose abdominal pains I did not properly appreciate on my first visit. I was called again a day or two later. The patient related that while she was having a bowel movement she was attacked by a severe pain in the lower abdomen. She was in an outdoor closet at the time and it was with the greatest difficulty that she made her way back to the house. At that visit a diagnosis of extra-uterine pregnancy was made. The patient was operated upon and the diagnosis confirmed.

A few days after the operation of patient number two, I called on patient number one for the purpose of reviewing the symptoms in her case. Upon questioning her, the fact was brought out that a day or two previous to her operation she was attacked by severe abdominal pains while at stool.

Since my experience with the two above cases, I have seen six additional cases of extra-uterine pregnancy, making a total of eight. Of these eight cases, six have had the symptom mentioned above.

The symptom is simply a severe, sharp, cutting pain in the lower abdomen while the patient is at stool and occurring, as near as can be ascertained, at the time when the abdominal muscles contract in assisting to empty the bowel.

I believe this symptom to be practically pathognomonic of extra-uterine pregnancy. I have found it in no other condition. Abscesses in relation to the rectum such as ischio-rectal abscess, abscesses resulting from infection of the tubes, large appendiceal abscess that crowds against the rectum and can be palpated by a finger in the rectum, produce no such symptoms. The pain that accompanies new growths from the rectum or pelvis or bladder, the pains of ureteral calculi or kinked ureter or twisted pedicals of cysts, do not resemble it at all. It does not occur in acute perforations of the stomach, intestines, gall-bladder or appendix. In fact, it occurs only in extra-uterine pregnancy.

This symptom does not occur in every case. However, if the patient is closely questioned I think the symptom will be found, in at least seventy-five per cent. of the cases, although I realize that my conclusions are being drawn from a series of cases that is far too small.

I have never seen this symptom mentioned in text-books. No mention is made of it by Dudley, Garrigues, Penrose, Kelley, Winter, or Ashton.

I have been unable to find any literature on the subject excepting Dr. Graham's reference to it in the Mayo Clinics.

Perhaps this symptom has been observed by all physicians. In fact, I have been waiting these six years for someone to report this symptom together with a series of cases far larger than my meager experience will permit. If such has been reported it has never been brought to my notice with the exception of the few words by Dr. Graham, mentioned above. If it is not common I hope that its observation in the future may be of some help. I will conclude by reporting but three of the eight cases, for the reason that the report of them all would be more or less of a repetition.

No. 1. I was called to see Mrs. E., aged 29, a mother of three children, all of whom were healthy except that one had been born with defective hearing. The patient had had two miscarriages and suffered frequently from severe headaches. Otherwise her history was negative. She complained of a rather moderate pain in the left pelvic region. Her last menstrual period had continued a day or two longer than usual. The general appearance of the patient was that of an healthy individual. Examination revealed a lacerated, rather weak perineum. There was some tenderness in the region of the left ovary. Nothing could be palpated. Owing to the tenderness in the pelvis, the examination was not satisfactory. I was unable to make a diagnosis. I was abliged to leave the city in a hurry, and during my absence of two days another physician was called in, as the patient had complained of more pain in the pelvis. Owing to the fact that the abdomen was very sensitive, the patient did not permit the physicians to make a very thorough pelvic examination. In consequence of this, the physician told the patient she was hysterical, and that he could do nothing for her. When I saw her the second time her pains had become more severe and upon questioning her she related that while she was straining at stool she was seized with a severe sharp pain deep in the lower part of her abdomen. So acute was this pain that it was difficult for her to return to her bed. The menstrual flow had not ceased. The patient felt a little exhausted. Owing to the tenderness of the pelvic region examination was again more or less unsatisfactory. However, I thought I felt a little more than the normal resistance in the culdesac. I advised an operation, which was undertaken immediately, and a rather large pelvic hemorrhage ensued.

No. 2. Mrs. D., aged 32, wife of a physician, had been ill for several days. The family history was not obtained. The husband had been tuberculous. Her personal history was not carefully elicited, but the scant information acquired proved that the patient had not been in the best of health. She had had two miscarriages and one child lived for only a few days. There were no living children.

The patient felt nauseous and had some pain in the lower abdomen and a severe lumbar ache. There was a little vaginal blood, but both the patient and her husband explained that little dependence could be placed upon this finding, as her menstruation was very irregular. I saw the patient at noon. During the forenoon her bowels had moved frequently, but passed only small amounts. She related that during one movement she had felt a sharp cutting pain deep in the pelvis, seemingly near the rectum. An abdominal and vaginal examination revealed nothing else other than tenderness. She was removed to a hospital immediately. In two hours after the first examination, and after a hypodermic had been given, a boggy mass could be felt in the culdesac. She was operated upon immediately.

No. 3. I saw the wife of an insurance man in consultation with two other physicians. Neither the family history nor personal history was obtained. On the second evening before my visit the patient had a pain of moderate severity in the right lower abdomen. Her family physician was called and he concluded that there was a little dietary disturbance. On the following evening he was called again, and he then found the patient in severe abdominal pain, mostly on the right side. The patient vomited and had a chill. She became rather weak and more or less thirsty. The physician remained with the patient all night. The next morning another physician was called, and at eleven o'clock I saw the patient with the other two physicians. Among other symptoms the patient stated that during the night previous, while she was at the stool shortly after vomiting, she had a severe pain in the lower abdomen, shooting through to the lower sacral region. A bimanual examination easily revealed a mass posterior to the cervix. She was operated upon immediately. An exceedingly large amount of clots was found in the lower abdomen.

DIFFERENTIAL DIAGNOSIS OF DIARRHEAS IN BOTTLE-FED INFANTS

BY ROGER HERBERT DENNETT
Adjunct Professor of Diseases of Children, New York Post-Graduate Medical School and Hospital
New York

The classification of diarrheas has never been upon a satisfactory basis, and since the treatment necessarily depends entirely upon the kind of diarrhea with which we are dealing, it is extremely important to make a correct diagnosis at the earliest possible moment—the dietetic treatment of one variety of diarrhea being exactly opposite to that of another kind. It is a great mistake to look upon all diarrheas of bottle-fed infants as infectious diarrheas since the majority of them, especially in very young babies, are due to intestinal indigestion.

Diarrheas may be divided into three classes—intestinal indigestion, infectious diarrhea, and miscellaneous. Intestinal indigestion is the etiological factor in the largest class of diarrheas. Infectious diarrheas are most often seen in the summer or in warm weather. However, the two may overlap each other since a baby with any form of indigestion is more susceptible to an infection and may, therefore, be more liable to an infectious diarrhea.

CLASSIFICATION OF DIARRHEAS IN BOTTLE-FED INFANTS

INTESTINAL INDIGESTION

(Dyspepsia, Lowered Tolerance for Food)

1. Simple intestinal indigestion from
    - fat
    - sugar
    - proteid
    - starch
2. Underfeeding, accompanied by intestinal indigestion.
3. Overfeeding, accompanied by intestinal indigestion.
4. Recurrent diarrhea, due to lowered tolerance that cannot be raised by the usual methods above a point where a proper and continuous gain in weight may be made.

INFECTIOUS DIARRHEAS

1. Fermentative diarrhea (dyspepsia, sugar or starch diarrhea, sugar intoxication).
2. Putrefactive diarrhea (proteid diarrhea).
3. Mild types of infectious diarrhea.
4. Dysentery (acute infectious diarrhea, ileocolitis, intoxication).
5. Cholera infantum.

MISCELLANEOUS

1. Sugar intoxication.
2. Mechanical diarrhea.
3. Diarrhea from cathartics.
4. Narasmus (lost power of assimilation, decomposition, atrophy).
5. Other organic causes (typhoid, amebic dysentery, tuberculosis, ulcer, intussusception).

Simple intestinal indigestion is caused by feeding mixtures containing more fat, sugar, proteid or starch than the individual infant is capable of digesting, or by feeding these elements in a form that cannot be digested by the infant. Most infants with diarrhea due to intestinal indigestion have a combination of fat, sugar and proteid indigestion which perhaps was originally due to one of these elements only. For instance, a diarrhea which was originally caused by fat may have so lowered the digestive capacity of the intestines that the power to digest fat, sugar, proteid or starch is decreased. Often it is not possible to differentiate between fat, sugar, proteid or starch diarrhea, nor is it necessary from the very practical standpoint of treatment, for, by omitting the starch and artificial sugar altogether, boiling the milk to make the proteid more digestible and cutting the fat down to the amount contained in about one-third milk and two-thirds water, the diarrhea may be overcome.

Indigestion caused by fat is usually due to the use of top milks or cream. These feedings do not necessarily cause indigestion in all infants. They may be easily digested by infants having greater digestive capacity or by those whose digestive power has not been lowered or overtaxed by such mixtures. The author believes that fat indigestion is seldom so severe that a baby cannot take all the fat contained in whole milk (mixed milk), diluted three or four times, provided no artificial sugar is added to it. It is occasionally necessary to use skimmed milk mixtures in extreme cases.

Sugar indigestion is caused by giving too much sugar. Some infants have a very low capacity for digesting sugar, often due to the error of giving too much of it at some time in the past. Often too much sugar is given at the beginning of artificial feeding or the sugar is later increased beyond the normal capacity of the infant. Sugar is frequently given too soon or increased too rapidly after an attack of diarrhea. The increase in an infant's tolerance for sugar is very gradual and consequently the sugar must be increased gradually.

Gruels are undoubtedly a contributing factor in the cause of diarrhea in very young infants when given in milk and sugar mixtures. For this reason and also to simplify the treatment of diarrhea by eliminating this possibility, gruels are best not used as dilutents in feeding these infants. Since it is not necessary, as was formerly thought, to use gruels to make the curds more digestible, nothing is gained by giving them, and one possible source of indigestion is eliminated.

The word "tolerance" seems a very good term to use as an expression of an infant's ability to digest and assimilate food. Indigestion, whether from sugar or fat, lowers the infant's tolerance for any food. Feeding weak dilutions of milk, without sugar, gives the gastrointestinal tract a chance to recuperate from the fat and sugar indigestion and thus raises the tolerance, so that the milk can gradually be made stronger, increased in quantity and sugar added. While these increases are being made in the food, the tolerance is increasing. Practically all cases of simple indigestion due to fat, sugar, or starch are amenable to this treatment, which is the reason the author has not deemed it necessary to classify them separately.

Underfeeding includes all those cases that have not had sufficient food to make them gain, even though they may not have had any indigestion. Unfortunately though, many of these babies have a more or less severe grade of intestinal indigestion and diarrhea also, and their tolerance had been gradually reduced throughout the time the indigestion has lasted. They are therefore a difficult class of cases to treat.

Underfeeding is usually brought about in the following manner:

an infant having a diarrhea due to intestinal indigestion is wrongly treated by weakening the food, that is, giving a mixture containing less fat, a little sugar and less proteid. This does not stop the diarrhea so the food value is again reduced. This weaker food is still undigested and the process of weakening is repeated until the infant is getting not nearly enough food to sustain it. In the same way an infant is wrongly given plain barley gruel to stop a fermentative diarrhea. The stools, of course, do not improve and the plain gruel is continued sometimes for two or three weeks because it is reasoned that if the baby cannot digest so simple a food as gruel, it would certainly not be able to digest milk. Diarrhea in such cases is due to starvation pure and simple. Possibly the fact that the gruel does not leave enough residue in the intestinal canal to enable the intestines to functionate properly also enters into the cause. Malted milk and some of the proprietary foods which are given without milk often act in the same way and, in addition, the diarrhea may also be fermentative in type on account of the high carbohydrate content of the food. Milk and water boiled together without sugar will correct the disturbance.

Overfeeding is easy to overcome if it occurs in well-nourished children. When occurring in poorly-nourished children, the ease with which the disease is remedied will depend upon the severity of the diarrhea. This subdivision needs little explanation. One frequently sees infants that are being fed much more food than they need and, if calories are reckoned, it is found that a fraction of the amount of food they are receiving would be sufficient. On the other hand, expert pediatricians occasionaly increase the food beyond an infant's needs in their enthusiasm to make a big gain in weight. This may bring about diarrhea with stools showing various degrees of intestinal indigestion. One-third milk and two-thirds water boiled together will also stop diarrhea due to overfeeding.

Lowered tolerance that cannot be raised by the usual methods above the point where the infant will make a gain in weight needs further explanation. Some babies have a very narrow limit between their digestive capacity and the amount of food they need to make a gain in weight. The indigestion can be overcome by stopping the sugar and giving boiled milk and water mixtures, but when the sugar is again added or the strength and quantity of the food is increased sufficient to permit the infant to make a gain in weight, a recurrence of the diarrhea is brought about. This may be repeated many times and is due to the fact that the infant's tolerance has been permanently lowered for these mixtures. The condition is frequently not recognized until repeated unsuccessful efforts have been made to increase the food to a sufficient strength. This is more apt to occur in an infant who has previously had many attacks or one prolonged attack of diarrhea.

The tolerance is lowered for one particular kind of sugar, or in certain instances for more than one kind. The diarrhea can usually be controlled by giving boiled milk and water mixtures or by Eiweiss Milch, and after the stools have become firm and smooth an entirely different kind of sugar should be given in extremely small amounts. As the tolerance increases the sugar should be increased with great care.

Differential Diagnosis of Simple Intestinal Indigestion. The diagnosis of simple intestinal indigestion presents few difficulties. The onset is invariably gradual, the infant going from bad to worse on a feeding that it is unable to digest. There is no fever, in fact a subnormal temperature is not uncommon in babies who are underfed or who are poorly nourished. During this period the weight is stationary, or there is a gradually progressive loss in weight. Occasionally the weight may show a slight increase, but a normal gain of from six to eight ounces a week is never possible where intestinal indigestion is marked. The cause may always be traced to a food which is unsuitable for the individual infant.

The stools may vary in number from two to ten or twelve in a day and an examination of them may show many abnormalities. They may be either yellow, green or brown in color. They may be watery or firm in consistency, but are rarely smooth and homogeneous. A smooth stool may at times show curds and a certain amount of mucus, and is indicative of intestinal indigestion. The odor of the stool is almost invariably normal and it is most important for the physician to be able to recognize the normal fecal odor of an infant's stool.

The reaction is not significant for it may be either acid or alkali, although it is more apt to be acid. Mucus is almost invariably present, in fact, if there is no mucus one may be certain that there is not much intestinal indigestion. Crying perhaps may be due to hunger rather than intestinal indigestion so that one may be sure there is no intestinal indigestion in a crying infant that has noraml stools.

Blood is never present in the stools of intestinal indigestion unless it is due to some local condition found in the rectum or anus, as a fissure or polypus. Curds usually are found in the stools of infants suffering from intestinal indigestion when milk has been given in the food. Infants who are being fed upon such foods as malted milk, or any foods given without milk, may have a marked attack of intestinal indigestion without showing curds in the stool.

Fermentative Diarrhea. Although great advances have been made during recent years, concerning the bacteriology of the intestinal tract, many questions still remain unsettled. It is known, however, that the intestines normally contain both putrefactive and ferementative bacteria as well as other varieties. Under the proper conditions, it is possible for the fermentative bacteria to be in the ascendency over the putrefactive bacteria and the reverse is also true. These proper conditions are brought about by supplying the food upon which the bacteria will thrive. If a carbohydrate diet is given the putrefactive bacteria, theoretically, are starved out. This forms a basis for treatment which from a practical standpoint is very satisfactory in its results.

The fermentative type of diarrhea is caused and aggravated by the bacteria which feed upon carbohydrates, that is starch or sugar. The Finkelstein school attributes this form of diarrhea to carbohydrate indigestion, Kendall has shown the bacteriological cause. Undoubtedly, many of these cases of diarrhea are due to sugar indigestion and many others to the growth in the intestinal canal of bacteria which live on carbohydrates. This is an academic question, however, which has no place here since the same treatment of stopping the carbohydrates and giving milk and water boiled together is effective whatever the cause. Sugar intoxication, according to the Finkelstein school, is due to the chemical action of the sugar, but there can be no doubt that, although this may be so in many instances, it is also due to the toxins generated by bacteria. The fermentative type of diarrhea is by far more common than the putrefactive type because of the universal habit of giving a large amount of sugar to bottle-fed infants. Another reason for this fact is that it has been asserted by various investigators that fermentative bacteria take precedence over putrefactive bacteria; or as Kendall expresses it, "Carbohydrate shields and spares protein from bacterial attack."

A bacteriological examination of the stools is not practicable for the general practitioner and will not be found necessary.

Putrefactive diarrhea is caused by the bacteria which fed upon protein. It is rather an uncommon type of diarrhea in bottle-fed infants except in cases where the method of omitting sugar and starch in the treatment of digestive disurbances has been followed. The reason for this has already been stated, namely, that fermentative bacteria tend to kill off putrefactive bacteria. In some cases there are evidently enough putrefactive bacteria left in the intestinal canal after the sugars have been eliminated from the food to cause a putrefactive diarrhea. In addition to this power of killing each other off, these bacteria seem to possess the power of changing themselves from fermentative to putrefactive organisms. In other words, in treating a case of fermentative diarrhea by stopping the sugar, one often sees a fermentative diarrhea change into the putrefactive type. When this occurs it is necessary to give a pure carbohydrate diet in order to starve out the putrefactive bacteria.

Differential Diagnosis of Fermentative and Putrefactive Diarrhea. It is often difficult to distinguish fermentative from putrefactive diarrhea. However, it is easy to distinguish either of the two from simple intestinal indigestion by the fact that a low grade of temperature, usually running from 99 deg. to 102 deg. F. is present in the fermentative and putrefactive types. The onset of fermentative diarrhea is more sudden than that of simple intestinal indigestion.

The stools of intestinal indigestion have no abnormal odor while those of the fermentative or putrefactive type are either sour or foul smelling. The consistency does not differ from simple intestinal indigestion, but, in fermentative diarrhea, we often see the foamy stool if it is observed when freshly passed. Unfortunately, the physician does not usually have the opportunity of seeing the stools until the foamy appearance has disappeared.

In differentiating fermentative and putrefactive diarrhea from the severe toxic forms of diarrhea like dysentery, cholera infantum and sugar intoxication there need not be much difficulty. In the latter types, the onset is always sudden, the temperature high (usually above 104 deg. and even as high as 106 deg. F.) and the infant always looks extremely ill and grows rapidly worse without proper treatment—and sometimes in spite of it. In dysentery of cholera infantum, the stools are apt to be more numerous, perhaps twenty in twenty-four hours. The stools of dysentery contain blood, and those of cholera infantum are entirely composed of mucus and serum.

In distinguishing between the fermentative and putrefactive type of diarrhea one finds much more difficulty. The onset is the same, usually sudden with a low degree of temperature—99 deg. to 102 deg. F.—and the infant looks only slightly ill. Apart from the stools there are only two main points of distinction. The fermentative diarrhea almost always occurs in the infant who has been fed a high sugar or starch diet, whereas the putrefactive diarrheas occur in infants who have been given strong milk mixtures with little or no starch in them, or in older children who are being fed on a mixed diet. For the reason that most formulas contain a great deal of sugar or starch, the majority of these diarrheas are fermentative in type, and especially so in infants under six or seven months of age.

The therapeutic test is the second means of distinguishing between the two. If one is in doubt whether he is dealing with a fermentative or putrefactive case, he may try a one-third milk and two-thirds water mixture (boiling together *without* sugar). If it is a fermentative diarrhea it will respond readily to this treatment. If it is the putrefactive diarrhea, the treatment will be of no avail and a starch diet, consisting of a thick gruel, will have to be used before the diarrhea is overcome. It is usually better when using this therapeutic test to give a *boiled* milk mixture to the younger infants, say under five or six months of age, and the gruels to infants over that age, since the younger infants are more liable to fermentative diarrhea and older ones to putrefactive diarrhea.

The stools of fermentative diarrhea are usually more numerous than those of putrefactive diarrhea. The color is not significant, nor is the presence of mucus or curds. The consistency, the typical foamy, watery stool of fermentative diarrhea differs greatly from that of the rather firm stool ordinarily seen in putrefactive diarrhea.

The reaction of the stool in fermentative diarrhea is always acid usually giving rise to inflamed buttocks and chafing. The reaction of the stools of putrefactive diarrhea is always alkaline and is not apt to be irritating to the skin.

In determining the reaction of the stool, it is necessary to get a stool that has been freshly passed and which is uncontaminated by urine. In order to do this, it is advisable to insert a hollow glass tube into the rectum taking out the fresh feces in this way for examination.

The sour odor of the stools accompanying fermentative diarrhea is very characteristic and the foul odor of putrefactive diarrhea (which resembles the odor of decayed meat) is also very helpful in differential diagnosis.

Dysentery (Synonymous with Ileocolitis, Infectious Diarrhea).— Up to the present time, four different microorganisms have been isolated from the stools of babies having dysentery. There are the Flexner dysentery bacillus, the Shiga bacillus, the colon bacillus and the streptococcus. According to Kendall, these bacteria no longer produce toxins when the diet is exclusively carbohydrate. This explains why the purely carbohydrate diet has long been successful in combating dysentery. There are the cases that have been variously called ileocolitis, dysentery, cholera infantum, and possibly some of the cases classified by Finkelstein as intoxication. There is a history of a sudden onset, high temperature, prostration, vomiting and extremely numerous loose stools composed of mucus and blood.

The diagnosis of dysentery presents few difficulties. An infant who has been perfectly well or one that has had intestinal indigestion is suddenly attacked with a very severe illness. The temperature ranges from 104 deg. to 106 deg. F., there is uncontrollable vomiting at the onset and a rapid loss of weight. The stools are very numerous, ten or twenty—or even more—in twenty-four hours and after the fecal matter which was in the bowels at the onset has been evacuated, the stools are composed almost entirely of blood and mucus, each evacuation being accompanied by a great deal of tenesmus.

Cholera Infantum.—It has been questioned by some authorities whether cholera infantum should be dignified by a classification of its own since as yet no bacteria have been isolated as its direct cause. It would probably be better to call this condition intoxication were it not for the fact that it might be confused with Finkelstein's sugar intoxication.

Clinically, cholera infantum has a very definite entity. It is characterized by a sudden onset, high rise in temperature, almost continuous vomiting, profuse diarrhea, stools which quickly lose their fecal character and are white in color like rice-water, and consist almost entirely of serum and mucus. All fluids of the body are rapidly drained, collapse follows, often with coma, and death within from twelve to thirty-six hours of the onset.

Sugar Intoxication.—Finkelstein and his co-workers, Meyer and Jerome S. Leopold, have proven conclusively that milk sugar can, of itself, produce symptoms of intoxication. They have brought on fever and symptoms of sugar intoxication by giving large amounts of sugar to children who were particularly vulnerable to its toxic effect, producing the so-called "Sugar Fever." Leopold and Reuss have experimentally produced "Sugar Fever" in young infants. The symptoms are fever, diarrhea and sometimes vomiting, deep slow respirations, stupor with a typical and characteristic facies, that is, the eyes sunken and staring, the mouth open and twitching. Sugar may also be recovered in the urine.

Marasmus (Lost power of assimilation, decomposition, atrophy). —This condition is so well known that it hardly needs an explanation. According to Grulee it is "a chronic state of malnutrition seen in infants, characterized by inability to so assimilate the food given as to gain weight properly, by subnormal temperature, emaciation, and by a greatly lowered resistance." The name "decomposition" has been given by Finkelstein to this well-known condition, but it only serves to confuse the nomenclature.

Diarrhea from Cathartics.—Diarrhea is frequently caused by the continued and daily use of cathartics, and it is very important to recognize this form in order to correct it.

Mechanical Diarrhea.—This class of diarrhea has grown smaller with our increased knowledge of the principles of infant feeding. It was formerly thought that the curds and various other constituents of the food acted mechanically only through irritation of the bowel and thus caused diarrhea. Mechanical diarrhea is now limited to cases occurring in infants and older children who have been given some specific indigestible article of food, such as bananas, undercooked starches, or the various other foods unfit for children.

This form of diarrhea has also been the cause of the absurd practice of treating all diarrheas with a cathartic and period of starvation. Such treatment is, of course, necessary in mechanical diarrhea.

Other Organic Causes.—Typhoid fever, amebic dysentery, tuberculosis, ulcer and intussusception are noted here only to make the classification complete. A discussion of them would be out of place in this paper.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOBLISM

Indicanemia and Hyperindicanemia in Persons with and without Kidney Disease—M. ROSENBERG, Münchener med. Wochenschr., Jan. 25, 1916.

The milder degrees of hyperindicanemia (which are only demonstrable by means of Jolles' test and not by the method of Obermeyer) cannot serve as indicators of renal insufficiency, as a similar degree of hyperindicanemia may occur in the blood of healthy persons and especially in other diseases without renal insufficiency. The border line at which hyperindicanemia positively indicates renal insufficiency is situated where the Obermeyer-Tschertkoff method demonstrates the presence of blood-indican. For this reason this method is to be preferred for the detection of blood-indican in clinical work. It is probably advisable to reserve the term hyperindicanemia for those cases whose blood-indican is demonstrable by this method. MILL.

Rate of Urea Excretion—T. ADDIS and C. K. WATANABE, Jour. Biologic. Chemistry, March, 1916.

The rate of urea excretion varies in man under physiological conditions in a manner which cannot be explained by the concentration of urea in the blood and urine. Other factors than urea concentration are important in determining the rate of urea excretion by the normal kidney. SACHS.

Ketones and Beta-Hydroxybutyric Acid in Urine of Normal Children—B. S. VEEDERS and M. R. JOHNSTON, Am. Jour. Dis. Child., April, 1916.

Authors always found small amounts of ketones and beta-hydroxybutyric acid in the urines of normal children, even when their diet was sufficient to cover their caloric requirements. The amount found varied from 20 to 100 mg. in terms of aceton in the twenty-four hours. Beta-hydroxybutyric acid is generally though not always, in excess of the ketones. When aceton bodies are in excess in the urines in febrile conditions and on restricted diets, they should be regarded as an increase of substances normally present.

SACHS.

Acidosis Occurring With Diarrhea—J. HOWLAND and W. MCK. MARRIOTT, Am. Jour. Dis. Child., May, 1916.

Acidosis is found in many cases of severe diarrhea not of the ileocolitis type. The clinical expression of the acidosis is hyperpnea. The presence of the acidosis has been confirmed by determining a lowering of the carbon dioxid tension of the alveolar air, by an increase in the hydrogen ion concentration of the blood serum, by a diminution of the alkali reserve of the serum, by an increase in the amount of alkali required to alter the reaction of the urine (alkali tolerance), and by a diminution of the combining power of the hemoglobin with oxygen. It has been shown that the administration of sodium bicarbonate will often bring about a cessation of the hyperpnea and cause the laboratory tests to give the results that are found with normal children. The acidosis is not due to the presence of aceton bodies. It has not been demonstrated that it is due to loss of base. It is probable that it is due to deficient excretion of acid phosphate by the kidneys. SACHS.

Acidosis of Chronic Nephritis—F. W. PEABODY, Arch. Int. Med., Dec., 1915.

The acidosis of chronic nephritis is the result of inefficient renal excretion. In uremia, acidosis is probably a very constant feature, but only in a small number of cases is it of sufficient grade to cause clinical symptoms similar to those seen in advanced cases of diabetes. In cases of chronic nephritis of a mild type, where the phenolsulphonephthalein test shows normal renal function, there is, as a rule, little or no acidosis. Advanced cases of chronic nephritis, in which the phenolsulphonephthalein output is moderately or extremely decreased, may show an acidosis by the "alkali tolerance" test, but the alveolar carbon dioxid tension may not be diminished. Only in very advanced cases is the acidosis usually so marked as to cause a decrease in the alveolar carbon dioxid tension. SACHS.

A New Blood Stain—B. LEMCHEN, Med. Rec., April 1, 1916.

The stain for blood smears consists of a saturated solution of benzidine in absolute alcohol. After making the blood smears on slides the solution is put on it for about half a minute. This fixes the blood on the slide and also impregnates the stain. Then hydrogen peroxid is added. After about half a minute the slide is washed in water and dried in filter paper. On examining the smears it will be found that the red cells are stained blue. Nucleated red cells, the cells together with the nucleus, are also stained blue. White cells do not take the stain. The blood platelets do not stain. If the blood smears are permitted to stand for some hours so that fibrin may form, the fibrin also stains blue. The conclusions to be drawn from this are that the red cells and white cells have different origin; that the platelets have not their origin in the nucleus of the red cells, and that the fibrin apparently has the same composition as the red cells. SACHS.

Oxydase Reaction on Blood Smears—F. A. EVANS, Arch. Int. Med., Dec., 1915.

In order to determine the origin of white blood cells of uncertain identity, Evans resorts to the demonstration of the presence or absence of oxydase granules in many varieties of white blood cells and so makes possible the differentiation between cells of myeloid and lymphoid origin. The blood smears should be as fresh as possible. They are fixed for from 6 to 8 hours in the vapor of 4 per cent. formaldehyde solution in a closed jar. They are then stained for eight minutes in a saturated aqueous safranin or a 2 per cent. pyronin solution, washed and dried immediately and examined in order to become familiar with the appearance of the various cell types with the nuclei thus stained. One drop of solution 1 (less then three weeks old) and one drop of solution 2 (less than four days old) are put on slide and the smear is mounted in this and examined at once, or the solutions may be put directly on the smear. Observe the reaction progress to its height by means of a low power objective and at the end of two minutes, dry and examine with the high dry objective. SACHS.

Leukocytes in Malignant Disease—O. C. GRUNER, Brit. Jour. Surg., Jan. 16, 1916.

The cell-findings in a blood smear vary in health under the influence of diet. The findings vary in disease according to the type of product of the causative agent, and not according to the agent itself. The findings in malignant disease vary according as the mass is actively growing or not, according to its limitation by fibrous tissues or not (no discharge of specific excretions in the juices) and according to the gross mechanical effect of the neoplasm on the body (starvation, hemorrhage). Monocytosis is a frequent accompaniment of protozoan infections. It is also decided in some cases of carcinoma and in many cases of sarcoma. Since one phase of the blood-picture produced by circulating toxins of malignant disease can be imitated by ingestion of highly nitrogenous food, it suggests that long continued over-use of the same may form an advantageous substratum for the subsequent development of the disease. While this statement has only speculative value, there is a practical value in the fact that blood smears should be taken before a meat meal, or preferably after instituting a purin-free diet for one or two days before. Author, from his investigations, formulates the following rules: If the smear shows neutrophilia, and fat drops in many of the neutrophiles; if the nuclei of the neutrophiles are largely multifid: the case is one of a coccal infection of great or

very great severity. If the smear shows a relative abundance of lymphocytes (especially of the very small variety); if there is no leukocytosis; if multifid nuclei preponderate; the case is almost certainly not one of maligant disease. If there is neutrophilia, with bizarre forms, or pseudopods in number; if the lymphocytes are in many instances showing ameboid outlines, if the monocytes show ameboid nuclei, the case is almost certainly one of maligant disease.
SACHS.

Detection of Albumin in Urine—LIEBERS, Deutsche med. Wochenschr., March 16, 1916.

Author finds that Pandy's reaction for the detection of albumin in urine is not only sensitive, but also very reliable. It is performed as follows: A ten per cent. solution of phenol is placed in a watch glass so that this is three-quarters filled. One or 2 drops of urine are permitted to drop into the phenol. In case albumin is present a whitish turbidity or a white cloudy deposit is formed. The latter may be dissolved by the addition of NaHO or NH3. MILL.

Glycosuria and Diabetes in Surgical Diseases—G. E. KONJETZNY and W. WEILAND, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXVIII, No. 5.

Examination of 83 instances of bone fractures. In nearly 50 per cent. of the examined cases spontaneous or alimentary transient glycosuria and hyperglycemia could be demonstrated. Authors consider the phenomenon to be of central origin. The phenomenon must be rigidly differentiated from traumatic diabetes. MILL.

Lipase and Lymphocytes—A RESCH, Deutsches Arch. f. klin. Medizin, Vol. CXVIII, No. 2.

The lymphocytes are the place of generation of the lipolytic ferment which is found in artificially produced exudates in serous cavities. This ferment has its optimal activity about the true neutral point and the H-ion- concentration of the blood. The mononuclear cells in the exudate are lymphocytes in morphologic respect.
WESTERN.

Palpation of Peripheral Glands and its Clinical Import in Children during the First Two Years of Life—C. COERPER, Monatschr. f. Kinderheilkunde, Vol. XIII, No. 10.

Children born at full term invariably have at birth palpable axillary glands. In seven months' babies palpable axillary glands are not found at birth. The number of palpable glands is not dependent upon the weight of the child. Babies brought up on mother's milk have generally fewer palpable glands than bottle-fed infants. Children affected with severe chronic nutritive disturbances show numerous palpable glandular groups. There is no chronic nutritive disturbance without numerous palpable glands. Lues and sepsis cause the production of many palpable glands. Tuberculosis, on the other hand, is the instigator of comparatively few glands that are palpable. Supraclavicular glands appearing in the first few months of life are not specific as far as tuberculosis is concerned.

MILL.

Carbohydrate Metabolism in Hyperthyroidism—H. R. GEYELIN, Arch. Int. Med., Dec., 1915.

Eighteen out of 27 cases of hyperthyroidism showed a hyperglycemia either after fasting or from 2½ to 3 hours after breakfast. All of these cases had alimentary or spontaneous glucosuria. A blood-sugar reading of 0.1 per cent. or over is to be considered a hyperglycemia and this condition occurs in 90 per cent. of the cases of hyperthyroidism studied by the author. Glucosuria is also as frequently observed in cases of hyperthyroidism. In the mild cases, fasting or post-prandial hyperglycemia and spontaneous glucosuria are usually absent, while alimentary hyperglycemia and glucosuria are not infrequently found.

SACHS.

Bruit Over the Eye Ball in Exophthalmic Goiter—D. RIESMAN, Jour. A.M.A., April 29, 1916.

A bruit over the eye ball in exophthalmic goiter has been observed by various clinicians, but this sign is not mentioned by systematic writers on exophthalmic goiter, or by text-books on diseases of the eye. The sound, when present, is easily elicited by placing the bowl of the stethoscope over the eye ball with the lids closed. The bruit, like other signs of exophthalmic goiter, is not constant, and the author has been unable to find it in several typical cases of the disease. It must not be confused with a more or less continuous sound or hum heard over the eye ball, which is evidently due to the muscular movements of the eyelid. The bruit is a rhythmic murmur, keeping time with the pulse.

SACHS.

Thyroid and Circulatory Changes Following Experimental Ligation of the Thyroid Vessels—H. NEUHOF, Jour. Med. Research, July, 1915.

Careful technic and asepsis are essential in experiments conducted to determine changes resulting from interference with the blood supply of the thyroid gland. Uniform changes do not follow ligation of the thyroid arteries or the veins. When present, they are generally not profound. Alterations in structure are more common and more marked after ligation of the thyroid veins than after ligation of the arteries. Following the former there is a decrease in the size of the acini; irregularity in their shape, increase of the interstitial connective tissue, and diminution in the amount of colloid. The end result, however (after three to six months), is an increase in size and coalescence of acini, scanty interfollicular connective tissue, and diminished size and weight of the gland. A reduction in size and generalized rounding in shape of the acini are the chief results of arterial ligation. The changes in one thyroid lobe, following ligation of its veins or arteries, may exist, to a lesser degree, in the unoperated lobe. The only symptom or sign referable to "hyperthyroidism," following interference with the circulation of the thyroid lobes, consists in an acceleration of the pulse, of several months' duration, after ligation of the thyroid veins, and retardation of the pulse, of several weeks' duration, after ligation of the arteries.

SACHS.

Cachexia of Pituitary Origin—M. SIMMONDS, Deutsche med. Wochenschr., Feb. 17, 1916.

Description of 2 cases of grave enduring progressive cachexia in which but one anatomic substrate, namely, the entire destruction of the pituitary gland could be demonstrated. This type of cachexia can therefore be traced to the pituitary destruction. MILL.

Influence of Pituitary Feeding Upon Growth and Sexual Development (An Experimental Study)—E. GOETSCH, Johns Hopkins Hosp., Bull., Feb., 1916.

Pituitary extract (anterior lobe), when fed to young rats, has a stimulating effect upon the growth of the animal and upon its sexual development and activity. Posterior lobe extract, when thus given, has a retarding influence. Ovarian extract (corpus luteum) has a stimulating influence upon the female, and a retarding influence upon the male, sexual development. WESTERN.

Pathogenesis of Quincke's Edema—P. NEUDA, Wiener klin. Wochenschr., Dec. 23, 1915.

Observations made on 5 cases of the affection. There are three cardinal symptoms of Quincke's edema: (1) Acute splenic tumor during the attack; (2) urobilinuria; (3) blood findings, as abnormally high color index during the attack and fluctuations in the number of erythrocytes, and their increased resistance. MILL.

INFECTIOUS DISEASES

Clinical and Bacteriological Observations in Epidemic Meningitis—H. SILBERGLEIT and K. v. ANGERER, Deutsche med. Wochenschr., Jan. 6, 1916.

Observations made in 7 cases. Sepsis was present in all of them. In 4 cases a mixed infection with other pus-producing organisms could be demonstrated. All the cases died despite serum treatment.

MILL.

Meningitis as Determined by Laboratory Examination—J. B. NEAL, Monthly Bull. Dep. of Health, City of N. Y., March, 1916.

The cases seen by the department of health, New York City, fall mainly into four groups: (1) Purulent meningitis (due to meningococcus, pneumococcus, streptococcus or the influenza bacillus; (2) tuberculous meningitis; (3) anterior poliomyelitis; (4) meningism, in pneumonia, infectious diseases, gastroenteritis, etc. Usually the spinal fluid is cloudy in the first group and clear in the other three. Very early, in an early case of epidemic meningitis, and in certain cases of poliomyelitis, the fluid is slightly cloudy. The value of lumbar puncture in meningism, tuberculous meningitis, and anterior poliomyelitis is twofold. It clears up the diagnosis and relieves the pressure. This latter is very important, especially in meningism, where it is usually followed by immediate and permanent subsidence of the meningeal symptoms. Of course there is as yet not specific treatment for anterior poliomyelitis or for tuberculous meningitis. Tuberculous meningitis has been invariably fatal. The differential diagnosis of these four groups would seem to be easy from the description in the text-books; and these points are familiar to everyone. However, cases are rarely typical, and the diagnosis is often entirely impossible without a lumbar puncture. A meningism, for instance, often gives all the clinical signs—stiffness of the neck, positive Macewen and Kernig, absence of pupillary and knee reflexes—of a well-advanced meningitis. An abortive poliomyelitis, with its sudden onset, high temperature, hypersensitiveness of the skin, irritability, stiffness of the neck, positive Macewen and Kernig, and exaggerated knee jerks, can be differentiated clinically with no degree of certainty from an early epidemic case. While in the later stages, a polioencephalitis, with its stupor, low and irregular temperature, and moderately marked meningeal signs, often resembles very closely a tuberculous meningitis, and the differentiation is made still more difficult by the similarity in fluids—both being increased in amount, usually clear, albumin and globulin increased, a fair reduction of Fehlings (the reductions in the polioencephalitis being usually better than in the tuberculous meningitis)—and an increase in cells, there are usually over 80 per cent. mononucleus. If tubercle bacilli are not found the differentiation is very difficult to make before the results of animal inoculation are known. The examination includes the cytology, bacteriology by smear and culture, the chemistry— albumen, globulin and reduction of Fehlings—and the results of animal inoculation in cases with clear fluids where tubercle bacilli are not found. The cells are increased in all fluids, except in those from cases of meningism, which of course show no inflammatory reaction. There is a preponderance of polymorphonuclear cells in the purulent forms of meningitis, of mononuclears in tuberculous meningitis, and in anterior poliomyelitis. In rare instances, there is a preponderance of polymorphonuclears in the more acute forms of tuberculous meningitis and in certain forms of polioencephalitis. The albumin and globulin are increased in all cases with inflammatory involvement of the meninges. Fehling's solution is well reduced by the fluid of meningism and usually in anterior poliomyelitis, also in early cases of tuberculous meningitis, and in mild and recovering cases of epidemic meningitis. It usually fails to be reduced in later cases of tuberculous meningitis, and at the height of the disease in severe cases of epidemic meningitis. The reduction of Fehlings has more value in prognosis than in diagnosis. WESTERN.

Etiology of Scarlet Fever—F. B. MALLORY and E. M. MEDLAR, Jour. Med. Research, March, 1916.

Authors have obtained a strongly gram-positive bacillus, in scarlet fever cases from the crypts of the tonsils, in the exudation on the surface of the tonsils, in slight depression and erosions in the squamous epithelium covering the tonsils, the pillars of the fauces, the soft palate including the uvula, and the root of the tongue, in the trachea and in the lungs. The bacilli in tissues and in direct smears from scarlet fever cases are slightly larger than the organisms in culture, but otherwise resemble them. The bacillus dies out quickly in the lesions, so that after the second or third day following the eruption it is difficult or impossible to demonstrate it, but it opens the way for streptococcus invasion and seems to favor its growth. SACHS.

Malaria, Diagnosis of—J. R. SNYDER, Calif. State Jour. Med., April, 1916.

In cases in which plasmodia cannot be demonstrated in the blood, author employes the urobilin test. The presence of urobilin in the urine is not specific for malaria, but in the author's series of cases of malarial involvement of the liver the urobilin was so frequently encountered that the urobilin test must be considered a valuable aid in the diagnosis of malaria. The technic of the test is simple. Add equal parts (2 to 3 cc.) of a saturated solution of zinc acetate in absolute alcohol to fresh urine. Shake. Then add two to three drops of a special Lugol's solution, made up as follows: I 1 cc.; KI 2 cc.; water 50 cc. Shake and filter. A fluorescence in the filtrate indicates a positive reaction. The author's conclusions are as follows: The test for urobilin is simple and inexpensive. It can be done by inexperienced laboratory workers. It is not specific for malaria. It is not constant in the early stages of acute malaria. It was present in 73 per cent. of cases tested after the disease had existed six days. It is of particular value in subacute and quiescent malaria. WESTERN.

Enteritis Complicating Measles—K. SECHER, Berliner klin. Wochenschr., March 6, 1916.

Enteritis complicating measles may appear in two forms: (1) A primary form depending upon the activity of the morbilli virus itself upon the bowel, appearing very early in the clinical picture often together with the eruption; (2) a form, secondary in character, due to infection by the intestinal bacterial flora and occurring late in the course of the disease. MILL.

Systemic Infection and Dysentery Bacilli—S. R. BRÜNAUER, Wiener klin. Wochenschr., Feb. 3, 1916.

A systemic infection with dysentery bacilli seems to occur oftener than is usually accepted. Of 54 cases of positively demonstrated dysentery 4 cases showed dysentery-bacteriuria and one case dysentery-bacteremia. MILL.

Guinea-Pig Test for the Virulence of Diphtheria Bacilli—J. A. KOLMER, S. S. MOODY, and E. L. MOSHAGE, Am. Jour. Dis. Child., April, 1916.

Animal inoculation tests have been found of great value in determining the virulence of diphtheria bacilli and virulence tests are the only practical and safe means of determining the possible harmfulness of diphtheria bacilli, especially so in the management of diphtheria bacillus "carriers." The virulence tests must be sufficiently delicate to detect low degrees of virulence. The subcutaneous injection of 4c. c. of a good 24 hours' culture diphtheria bacilli on a tube of Loeffler's medium washed off with 10 cc. of normal salt solution, was found as delicate and specific in its results as any method, besides being a test requiring a short space of time for its conduct. Another method yielding excellent results, but requiring more time, consists in cultivating the bacilli in serum dextrose broth for 72 hours and injecting pigs subcutaneously with 1.3 to 1.5 cc. of unfiltered culture. Local edema and even slight evidences of toxemia of the test animal with absence of those signs in the antitoxin control, are regarded as indicating virulence even though the animal does not succumb in the four-day period of observation. SACHS.

Schick Test—D. M. GRISWOLD, Jour. Labor. and Clin. Med., March, 1916.

The following summary is offered by author: The Schick test seems to give a quick, easy way of determining whether a person is susceptible to diphtheria. Its use will eliminate needless giving of antitoxin to persons exposed to this disease. It will lessen the danger of anaphylaxis in a community because there will be fewer sensitized persons. It is the greatest single step forward in immunology and serum therapy since the advent of diphtheria antitoxin itself. WESTERN.

Schick Test—A. ZINGHER, Am. Jour. Dis. Child., April, 1916.

The results with the test obtained in 2,700 normal children shows that from 17 to 32 per cent. between the ages of 2 and 16 years give a positive reaction and are probably susceptible to diphtheria. The positive reaction should be considered as indicating a lack of immunity, unless the pseudoreaction can be eliminated by a control test. The negative reaction is a definite sign of immunity. Diphtheria toxin is an active reagent, but it will deteriorate, even in bulk, if it is not properly protected from light and exposure, and kept in a very cold place. SACHS.

Bacteria Associated with Abnormal Lymph Glands—J. C. TORREY, Jour. Med. Research, March, 1916.

A diphtheroid bacillus of one type or another was recovered from 22 of 30 cases in which the primary seat of the disease was in the lymphatic gland and of 10 cases in which the lymphatic glands were secondarily involved. Positive cultures were obtained in cases of Hodgkins' disease, chronic hyperplastic lymphangitis of obscure nature, lymphosarcoma, sarcoma, melanoma, endothelioma, tuberculous adenitis and chronic lymphatic leukemia. A number of other aerobic bacteria were found in these lymph glands. With the exception of staphylococcus, anaerobic bacteria were only sporadically present. SACHS.

Agglutination Tests in Inoculated Persons—G. DREYER, A. G. GIBSON and E. W. A. WALKER, Lancet, April 8, 1916.

Authors in their conclusions do not agree with those of Tidy. They claim that the febrile condition associated with paratyphoid does not cause the disappearance of typhoid agglutinins from the blood of persons inoculated against typhoid. Other febrile conditions also, in so far as they have hitherto presented themselves, do not cause disappearance of typhoid agglutinins from the blood of inoculated persons. They state that the assertion that a positive agglutination reaction to B. typhosus after the fifth day of pyrexia has the same value in an inoculated as in an uninoculated person is entirely erroneous. SACHS.

Search for Typhoid Carriers—A. STOKES and C. CLARKE, Lancet, March 11, 1916.

In a search for typhoid carriers authors examined 810 cases and found that 32 of these cases were of the temporary intestinal variety, and 33 were of the temporary urinary variety, while 11 were of the chronic intestinal type and 2 of the chronic urinary type. SACHS.

Fatal Cases of Paratyphoid Fever—B. DAMSON and F. H. WITINGTON, Quart. Jour. Med. (London), Jan., 1916.

Fatal cases of paratyphoid fever are sufficiently uncommon to be worthy of record. Authors base their paper on 2 cases of paratyphoid fever A and 15 cases of paratyphoid B with their postmortem appearances. Although in general, paratyphoid is a reduced image of typhoid fever, the occurrence of severe and fatal cases show that it has potentialities for greater mischief, and that under conditions favoring it, the disease might break out in severe epidemic form. The severe cases resemble typhoid fever in their clinical entity and the diagnosis must be made on the laboratory findings. Abdominal pain and diarrhea were the most frequent onset symptoms. The tongue resembles that of typhoid fever. In 13 cases spots were present and in some instances they were of larger size and less regular outline than those of true typhoid. Abdominal distension is often noticeable by its absence. SACHS.

Diagnosis of Typhus Fever—A. STRASSER, Zeitschr. f. physikalische u. diätetische Therapie, 1915, No. 11.

The following are the cardinal symptoms of the infection: The attack-like appearance of an eruption resembling that of measles. (This eruption may also appear upon the extremities; the eruption assumes the form of petechiae.) The marked splenic tumor disappears while the patient is still in the febrile stage. A high degree of intensity which disappears during the febrile period. Uniform grayish coating of tongue. Marked psychic phenomena. MILL.

Rocky Mountain Spotted Fever—L. PAINE, Northwest Medicine, Jan., 1916.

Differential diagnosis. The only disease with which Rocky Mountain spotted fever could be confused is typhus fever, but a knowledge of the nature of infection (the wood tick in Rocky Mountain spotted fever, and the body louse in unsanitary, poverty-stricken quarters in typhus), and the geographic distribution of the disease. Rocky Mountain spotted fever occurs in all the Rocky Mountain states, except Arizona and New Mexico, whereas typhus, on the western hemisphere, is most frequent in Mexico, and to some extent in some of the large eastern cities, New York, for example. WESTERN.

Comparative Sputum Examinations by Means of the Ziehl-Neelson and the Kronberger Methods—F. LEICHTWEISS, Zeitschr. f. Tuberkulose, Vol. XXV, No. 2.

Author demonstrates the superiority of Kronberger's carbolfuchsin-iodin stain over the Ziehl-Neelson staining method. Kronberger's stain should replace the older method. FRY.

Permanganate (Urochromogen) Reaction and Diazo Reaction—S. BOSCH, Deutsche med. Wochenschr., Jan. 6, 1916.

Ehrlich's diazo reaction, in author's experience, was only positive when the permanganate reaction was likewise positive. In grave cases of tuberculosis a positive parmanganate reaction ensued twice as frequently as a positive diazo reaction. In prognostic respects both reactions are of about equal value, viz., 100 per cent. of the tuberculous patients with positive diazo reaction and 96 per cent. of the same with positive permanganate reaction died within 8 months. In some patients the permanganate reaction was only positive at certain periods. MILL.

Fever: Initial Sign of Tuberculosis Infection in Infants—M. S. REUBEN, Arch. Pediat., March, 1916.

Author studied 9 cases which showed that fever is the first sign of tuberculosis infection in infants. The onset of the fever is sudden and is at first high. It is remittent and comes down by lysis, but not to normal. It may be 100 to 101 deg. F. for 12 to 14 weeks. There are periods of absence of fever and some exacerbations of high fever. While there are no physical signs at the beginning, within 2 or 3 weeks, these cases develop signs in the chest, which dissapear if the child gets well. The tuberculosis reaction usually becomes positive either at the onset of the fever or within a few days. Thirty to thirty-five per cent. of cases of tuberculous infection in infancy recover. SACHS.

Incipient Apical Pulmonary Tuberculosis—M. GROSSMAN, Am. Med., April, 1916.

One of the earliest signs in incipient apical pulmonary tuberculosis is the presence of a soft systolic murmur heard at the cardiac apex. Accompanying this murmur is congestion of various degrees heard with the stethoscope over the apex of the lung, and generally the left one. If this murmur is functional in character, then the condition is pulmonary tuberculosis, for localized congestion of an apex under such circumstances means tuberculosis of that apex. If, on the other hand, the murmur is organic, that is, a mitral insufficiency, then the congestion of the apex is, as a rule, not tuberculous, but is the result of the murmur. When a patient has a mitral insufficiency, he very rarely has pulmonary tuberculosis. The reason for this is that probably the tubercle bacilli will not find the apex a good soil for growth, as there is too lively a circulation of blood in this region on account of the congestion due to a murmur. If both pulses diminish or are absent when arms are held in upright position compared to arms held in horizontal position, in a majority of the cases, the trouble is one of mitral insufficiency, because the diminution of pulses shows weakened myocardium. If only one pulse diminishes or is absent, we may suspect, as a rule, tuberculosis of the apex corresponding to the pulse, this phenomenon being probably due to pressure effects of an incipient pulmonary tuberculosis. WESTERN.

Eye Findings in Pulmonary Tuberculosis—A. J. BEDELL, N. Y. State Med. Jour., April, 1916.

Author records the eye findings in 100 patients with pulmonary tuberculosis. The results showed: Hypermetropic eyes, 54; myopic, 3; myopic astigmatism, 12; mixed astigmatism, 7; hypermetropic astigmatism, 26; compound hypermetropic astigmatism, 39; emmetropic, 4. The pupils were equal in 69 and unequal in 31. This is a symptom to which attention has been drawn by numerous observers, but as yet the author is not convinced that it is of clinical value. In support of the latter statement, Bedell says that out of 100 patients presenting themselves in his office for refractive errors, 27 had unequal pupils. SACHS.

D'Espine's Sign in Childhood—J. L. MORSE, Am. Jour. Dis. Child, April, 1916.

Author concludes that D'Espine was correct in his original contention that the normal change in the voice occurs between the seventh cervical and first dorsal spines. D'Espine's sign is present therefore when the bronchial voice, or whisper is heard below the seventh cervical spine. When this sign is present in children of the well-to-to classes, it is probably not a manifestation of tuberculosis in more than 50 per cent. of the cases. SACHS.

Tuberculosis of the Mammary Gland—GATEWOOD, Interstate Med. Jour., April, 1916.

Tuberculosis of the breast must be differentiated from actinomycosis and syphilis. These lesions are rarer than tuberculous mastitis, and a diagnosis can readily be made on the hand of a careful history and modern methods of laboratory examination. If, however, the diagnosis is not entirely cleared up, the effect of antisyphilitic treatment should make it reasonably certain within a short time, as potassium iodid is almost specific for either lesion. Differentiation from chronic mastitis is not always so easy, and removal of tissue, which is after all the only absolute method of making a diagnosis, may be necessary. The sclerosing type or the discrete nodular type in which the process has become latent, without fistula formation, may give all the symptoms of mammary carcinoma and defy clinical differentiation. The following conclusions are drawn: Tuberculosis of the mammary gland is very uncommon considering the frequency of tuberculosis. The term "primary tuberculous mastitis" should be applied only to those cases in which there is definite evidence that the infection was introduced directly into the breast tissues. The symptoms are varied and the diagnosis should be made certain by the aid of the microscope before radical operations are undertaken. The pathology, although varied, does not differ essentially from that of tuberculosis in other organs. The treatment is surgical in almost every instance, but the postoperative care of the patient is very essential to complete recovery. The prognosis is excellent provided the general condition of the patient is good.

WESTERN.

Serology and the Diagnosis of Syphilis—M. L. HEIDINGSFELD, N. Y. Med. Jour., April 8, 1916.

Author draws the following conclusions: (1) The serological examination in syphilis is trustworthy if carefully and scientifically performed. (2) The serological examination can never err on the unsafe side, namely, be positive in the absence of syphilis, unless through technical error. (3) A serological examination may be negative and syphilis present in clinically active or latent form. (4) Both Wassermann and Hecht-Weinberg-Gradwohl tests are trustworthy. When combined they are a check to each other. The Wassermann reveals the degree of early improvement. The Hecht-Weinberg-Gradwohl is usually positive long after the Wassermann is negative, and measures with much greater delicacy and precision the last traces of serological inhibition. (5) Serology is a mere aid to clinical diagnosis and therapeutics, and as such belongs strictly in the province of the clinician of syphilis. It is indispensable to the clinician of syphilis who successfully essays the diagnosis and treatment of this affection. (6) A serological report must either be negative or positive; inhibition is either present or absent, and there is no place for a report "doubtfully positive" in character. (7) The diagnosis of dermatoses of doubtful nature or symptoms of uncertain character cannot be intrusted solely to serological evidence. Nonsyphilitic dermatoses, resembling syphilis, may possess an associated syphilitic infection, which a positive serological report will invariably cloud with greater obscurity. (8) A negative serological report in positive clinical syphilis in mystifying and may be attended with gravely deleterious results. (9) Many laboratory serologists require a personal examination of the patient or clinical description of the case. In either event, they pose as clinicians as well as serologists, and unless clinically well qualified, biased laboratory findings may plunge them into serious error. (10) Serologists who stamp a distinctly nonsyphilitic affection syphilitic, in individuals in whom the disease is entirely absent, commit a grave and inexcusable error. (11) Serology, in its limited scope, has for the time being usurped the broader province of the clinician. Frequent and repeated laboratory errors, coupled with erroneous clinical interpretations, demand the reinstatement of the old order of things. (12) Clinical evidence, today, as ever, takes precedence over laboratory findings. The serologist must again yield to the syphilographer, whose prerogatives, for the time being, have been unduly usurped. Questions involving doubt in clinical diagnosis and treatment in syphilis should find their proper solution, not in the hands of the serologist, but the dermatologist, syphilologist, neurologist, internist, oculist, etc., as the case may be, who in turn can call the serologist to his aid if occasion requires. SACHS.

Luetin Reaction in Congenital Syphilis—M. B. GORDON, Arch. Pediat., March, 1916.

Author is of the opinion that the luetin reaction is more applicable in pediatric work than the Wassermann test, especially is this so in dispensary work. The reaction was present in 81 per cent. of the cases of congenital syphilis studied by him and was absent in all but one of the non-syphilitic cases. SACHS.

Hereditary Syphilis—LE G. KERR, N. Y. State Med. Jour., March, 1916.

When inquiring into the family history as to the existence of hereditary syphilis in a child, it is never permissible to ask if any of the family have or have had syphilis, for this question will put the parents on their guard and will often elicit a denial, either intentionally or unintentionally. It is better to obtain a history of events which will establish strong presumptive evidence as to the existence of the disease. The events to be considered are these: (a) The tendency of the children of a given family to suffer from unaccountable anemias and malnutrition during the earlier periods of life, despite the fact that the hygienic surroundings and the dietetic care are both excellent; (b) the occurrence of anomalous types of disease in two or more children of the same family, or the unusual course of a disease under similar circumstances; (c) tardy development without a recognized cause, or occurring as the result of a recognized etiology, but out of all proportion to that cause, and occurring in two or more children of the same family; (d) the occurrence of a rachitic type of skull without other evidences of rachitis being proportionately marked; (e) history of the disease, direct or presumptive, in one or both parents; (f) a history of the abortive habit in the mother without definite cause. In making a prognosis in a case of congenital syphilis, it must be remembered that it is a much more fatal disease than the acquired form of adults. The earlier the symptoms appear, the worse the prognosis. SACHS.

Syphilis, with Secondary Involvement of Heart and Kidneys—A. EUSTIS, New Orleans Med. and Surg. Jour., April, 1916.

Patient was 12 years old and came to the hospital in a convulsion. Family history negative; no history to account for syphilis. Three weeks ago patient noticed sores on limb, and later swelling of lids and ankles, and later convulsions with a duration of about five minutes. Among the other symptoms were chills, nausea, coma, and general edema of both lungs, a presystolic murmur, ascites, anasarca.

Urine showed 15 per cent. albumin. First diagnosis entertained was one of uremia and endocarditis. The patient recovered. His urine became normal. WESTERN.

Acute Syphilitic Nephritis—J. H. STOKES, Jour. A.M.A., April 15, 1916.

Syphilis as a causative factor in acute nephritis is easily overlooked, or its significance is not appreciated. It may develop in the course of efficient mercurial treatment. The best recognized points in its diagnosis are: (a) Establishment of the existence of early syphilis; (b) high albumen content of the urine; (c) double-refractive lipoids in the urine; (d) therapeutic tests. Spirochæta pallida should be searched for in the sediment of a catherized specimen, as recommended by Hoffmann. The administration of salvarsan is a valuable aid in the diagnosis and treatment of acute syphilitic nephritis. SACHS.

RESPIRATORY AND CIRCULATORY ORGANS

Spontaneous Pneumothorax—L. HAMMON, Am. Jour. Med. Sci., Feb., 1916.

Author cites 5 cases of spontaneous pneumothorax which illustrate the insidious onset of some of these cases; the few symptoms this condition may produce; the perfectly benign course of the affection, and the occurrence of an insidious benign type of pneumothorax in perfectly healthy individuals as well as in those with manifest pulmonary disease. SACHS.

Pyorrhea Alveolaris, Chronic Bronchitis and Asthma—S. P. SHIRDAS, Practitioner (London), April, 1916.

Author states that pyorrhea alveolaris alone forms one of the potent etiological factors in the causation of chronic bronchitits and asthma. Cases of chronic bronchitis and asthma which were associated with a pyorrhea alveolaris were treated for the latter condition with splendid results. In those cases in which the pyorrhea alone was treated, either a marked improvement or disappearance of the lung affection was observed, with the arrest or improvement of the pyorrhea. When the affections alone were treated without any attention to the pyorrhea, hardly any good results were obtained, except a slight temporary relief in the symptoms, in consequence of the administration of soothing and anodyne remedies. When both the pyorrhea and the lung affections were treated, the asthma or chronic bronchitis either disappeared or showed improvement with arrest or improvement of the pyorrhea. SACHS.

Syphilis and Aorta—M. HERZ, Wiener klin. Wochenschr., March 9, 1916.

Among 7,000 cases of heart disease (changes of the aorta in the presence of lues) insufficiency was found 96 times, and aneurism 26 times. Syphilis without aortitis occurred 122 times, and dilatation of the aorta without lues was demonstrated in 249 cases. Insufficiency of the aorta without lues was found in 251 patients. The percentage of cases in the female sex, so far as luetic aortitis is concerned, was found to be 2.5. MILL.

Syphilitic Aortitis—J. H. LARKIN and I. J. LEVY, N. Y. State Jour. Med., Dec., 1915.

Authors examined 42 aortas and tested the blood of these patients serologically before death. Nineteen of these gave a positive Wassermann reaction. Seventeen aortas out of these 19 cases gave microscopic evidence of syphilitic aortitis. Fifteen of the 19 patients died as a result of a syphilitic process and 11 of these died from their syphilitic aortitis. Authors are of the opinion that it is probable that 90 per cent. of individuals dying with a positive Wassermann reaction in the blood, if the infection is not of recent origin, have a syphilitic aortitis. SACHS.

Rheumatic Heart Block—T. v. PODMANICZY, Zeitschr. f. klin. Medizin, Vol. LXXXII, Nos. 1 and 2.

Report of 2 cases which had probably arisen on the basis of a rheumatic myocarditis. Case 1. Soldier, 22 years old. Following an attack of acute articular rheumatism there occurred a heart block which lasted one day. During the heart block an attack of vertigo ensued. The heart block did not reoccur when large doses of digalen and strophantin were administered in order to slacken the pulse. Case 2. Male, 29 years old. Had experienced a number of attacks of articular rheumatism with involvement of the heart. A total and permanent heart block had developed of late. Sphygmographic and X-ray examinations confirmed the diagnosis. WESTERN.

Slight Enlargement of the Heart—G. C. SHATTUCK, Boston Med. and Surg. Jour., March 16, 1916.

Slight enlargement of the heart may be detected by a fluoroscope when a careful history and a skilful physical examination are inconclusive. In other cases an orthodiagram or a teleoröntgenogram may reveal a slight abnormality on the outline or measurements of the heart. Enlargements should be estimated by centimeters not by millimeters, as normal hearts are frequently variable in size. SACHS.

Soldiers' Heart—J. MACKENZIE, Brit. Med. Jour., Jan. 22, 1916.

Mackenzie states that at least 90 per cent. of cardiac cases in soldiers that have been certified and treated as heart affections are not primarily heart cases and that the treatment of these cases is very different from cases of true heart failure. The chief symptoms are the absence of a feeling of well-being, exhaustion and breathlessness on slight exertion and in a few cases, pain over the cardiac region. Variable physical signs may be present. The pulse-rate may or may not be increased, while in some cases slight exercise unduly increases it. A slight increase of the heart in size and systolic murmurs are frequently observed. Depression and irritability are not uncommon mental accompaniments. Treatment should be in the line of increasing the health of the body as a whole by fresh air and exercise. SACHS.

Heart's Action in Jaundice—J. D. WINDLE, Brit. Med. Jour., Jan. 22, 1916.

Author investigated the heart in a number of jaundiced patients and he is not of the opinion that the occasional irregularity or infrequent pulse is due to the jaundice. A pulse of 50 to 55 is frequently found in jaundiced patients who are in bed. Slowing of the pulse to 40 or below whether it results from disease or poison is in the vast majority of cases due to partial or complete heart block. SACHS.

ALIMENTARY TRACT

Gluzinski's Method in the Diagnosis of Gastric Disease—H. ZOEPPRITZ, Mitteilungen a. d. Grenzgebieten d. Medizinu Chirurgie, Vol. XXIX, No. 1.

Gluzinski's method was employed in 462 cases. So-called relative secretory insufficiency occurs comparatively often in the presence of gastric carcinoma and HCl, but may also be observed in other affections of the stomach and even when the stomach is healthy. The method is useless in the differential diagnosis between stenotic and non-stenotic carcinoma and between carcinoma and ulcer, and in the early diagnosis of malignant disease. MILL.

Acidity Determination of Gastric Juice—SEIDL, Archiv f. Verdauungskrankheiten, Vol. XXI, Nos. 2 and 3.

The gastric contents obtained after a test-breakfast possess, as a rule, a higher degree of acidity than the filtrate. The higher acidity degree is caused by a mechanical combination of the acid with the starchy material. Gastric mucus has the property of binding much alkali. For this reason acidimetry in the presence of considerable admixture of mucus with the gastric contents should be performed with the filtrate only. WESTERN.

Quantitative Acidity Determination of the Filtrated and Unfiltrated Gastric Juice—WEINBERG, Archiv f. Verdauungskrankheiten, Vol. XXI, No. 4.

Titration of the unfiltered gastric juice give more reliable results than titration of the filtered specimen. If possible, titration of both filtered and unfiltered gastric contents should be made in order to obtain data concerning the distribution of the acid contents.

WESTERN.

Superior Mesenteric Vessels in Abdominal Disease—J. N. HALL, N. Y. State Med. Jour., March, 1916.

These vessels, supplying a most important portion of the digestive tract, play a leading part in such a variety of conditions as to deserve special study from the internist as well as the surgeon. The superior mesenteric vein carries the return circulation from the appendix and cecum and many of its diseases originate in the septic processes from these organs or from their territory. The right gastro-epiploic vein drains the greater curvature of the stomach, and the pancreatico-duodenal vein supplies a region frequently involved in digestive diseases, and therefore afford opportunity for thrombosis and septic invasion. In patients of the arterio-sclerotic age, painful abdominal affections may well be due to spasm of the gastric and mesenteric vessels, and it is believed by many clinicians that angina abdominis is definitely due to such spasms. About twice as many cases of intestinal infarction occur in men as in women, generally in middle life. Arterial embolism usually results from an endocarditis or from an atheromatous degeneration of the aorta. Thrombophlebitis is usually secondary, occurring as an infective thrombophlebitis. The symptoms of intestinal infarction are acute in the majority of cases, although a chronic course is not infrequent. The most frequent syndrome is one suggesting acute obstruction of the bowel, with colicky pain in 96 per cent. of the cases, nausea and vomiting in most instances, and in 19 per cent. bloody diarrhea, not explicable by local disease of the bowel nor by portal obstruction. No dependence can be placed on the temperature, as fever is frequently absent. Acute dilatation of the stomach, originating from pressure upon the duodenum by the superior mesenteric vessels, is evidenced by continuous vomiting, pain, collapse, anuria, great thirst and constipation. The vomitus is brownish from capillary hemorrhage and from the bile which enters the stomach because of the blocking below its point of entrance into the duodenum. SACHS.

End Results of Operation on Perforated Gastric and Duodenal Ulcers—C. L. GIBSON, Surg. Gynecol. and Obstet., April, 1916.

When patients are operated on within 2 to 4 hours after the perforation of a gastric or duodenal ulcer, the mortality should not be more than 5 per cent. Thirteen cases which were operated on were observed for varying lengths of time. The perforation itself has a tendency to cure the ulcer and so it is unnecessary to do in most of the cases either a resection or a gastroenterostomy. Every one of the 13 cases observed after operation showed an improvement in general health and a relief or cure of previous gastric symptoms. X-ray examination of 12 cases showed that 7 of these had no retention at all, while only one showed a considerable amount of retained bismuth, which in all probablity was due to a ptosed stomach. SACHS.

Occult Hemorrhages in Ulcer and Carcinoma of the Stomach—I. BOAS, Archiv f. Verdauungskrankheiten, Vol. XXI, Nos. 2 and 3.

Though there is a superficial resemblance between the occult hemorrhages due to ulcer and cancer of the stomach, there is, nevertheless, a great difference (in form as well as in manner) between them. The hemorrhage in carcinoma is persistent, in ulcer occult bleedings cease quickly. It is absolutely necessary that not only the feces, but also the gastric contents be examined for occult blood. WESTERN.

Occult Blood in Gastric Contents and Feces in the Diagnosis of Gastric Carcinoma—J. EMMERT, Münchener med. Wochenschr., March 7, 1916.

Observations in 21 patients. In these cases the positive blood findings exceeded in frequency all other symptoms. The findings were negative in one case only. This symptom appears very early in the course of the affection and is probably already present when the patient looks for medical assistance. The test was already positive in one of author's cases in which gastric difficulty was only present for 4 weeks. The demonstration of blood in gastric contents may exclude a possible intestinal tumor. A positive guaiac test is characteristic of the presence of gastric carcinoma, and a single negative result of this test in all probability indicates the absence of malignant disease. MILL.

Salomon's Albumin Determination in the Gastric Contents—H. ZOEPPRITZ, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXVIII, No. 5.

Report of the examination of 148 cases (98 operated upon) by means of the Salomon albumin determination. The test has but a conditional value. As far as its practical value in the diagnosis of gastric carcinoma is concerned it occupies fourth place. A negative result of the test in all probability contraindicates the presence of malignant disease. Albumin is also occasionally demonstrable in the gastric contents of a clinically sound stomach. MILL.

Diagnosis of Duodenal Ulcer—O. WULFF, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 1.

Differential diagnostic observations upon 118 instances of duodenal and gastric ulcers that have come to operation. Author concludes that neither the Bucquoi-Moynihan symptom-complex nor the Hartmann-Souppault pyloric syndrome are of decisive value in differential diagnosis, and that the topographic diagnosis of an ulcer is a rather uncertain undertaking. MILL.

Gastric Carcinoma—SCHÜTZ, Archiv f. Verdauungskrankheiten, Vol. XXI, No. 6.

The assumption that gastric ulcer is frequently transformed into gastric carcinoma is by no means definitely demonstrated. In 222 cases of the author free HCl was absent in 90 per cent. And absolutely reliable sign of gastric carcinoma, lactic acid, was present in only 33 per cent. of the cases. In 88 per cent. of the cases a positive diagnosis could be arrived at clinically, i. e., without the results obtaining by röntgenology. In 16 cases, however, the X-ray examination alone revealed the nature of the affection, but in these cases scirrhus forms of malignancy were encountered in almost every instance. WESTERN.

Syphilis of the Stomach—M. EINHORN, Archiv f. Verdauungskrankheiten, Vol. XXI, Nos. 2 and 3.

Except by a microscopic examination of carcinomatous tissue there is no characteristic sign or symptom of gastric carcinoma which may not with equal certainty (or uncertainty) point to gumma of the stomach. An antiluetic therapy is often necessary in order to arrive at a positive differential diagnosis. WESTERN.

Etiology of Appendicitis—F. SAMBERGER, Wiener med. Wochenschr., Jan. 3, 1916.

Stagnation of the intestinal contents is supposed by many to be an important cause of appendicitis. This stagnation is favored by lying upon the right side. This posture is habitually preferred by the majority of people on account of a variety of circumstances. Special observations in persons with appendiceal disease point to the importance of aforementioned factors in the production of the pathological condition. Of anatomical import is the fact that there is a kind of valve-formation about the left flexure of the colon and that the rather rigid fixation by the ligamentum phrenico-colicum adds to the difficulty of transporting the feces into the descending portion of the colon. The transmission of the feces is facilitated by lying on the left side; it is rendered more difficult by assuming the right posture. MILL.

Gastric Chemism in Affections of the Gall-Bladder—OHLY, Archiv f. Verdauungskrankheiten, Vol. XXI, Nos. 2 and 3.

The majority of instances of chronic cholelithiasis show subacidity or anacidity. In 87 examined cases, HCl was 46 times either deficient in amount or absent. Hyperacidity of the stomach, on the other hand, was met with in 19 rather acute cases. The first attack of biliary colic frequently ensues in women during or after pregnancy. In other instances gastrointestinal affections overshadow the gall-bladder symptoms for years and years. Again in other cases obesity, metabolic disorders as diabetes and gout appear to be of etiologic import. WESTERN.

Cholelithiasis—C. C. MAPES, Am. Jour. Surg., Feb., 1916.

Author advances the following conclusions: (1) That there are many uncertainties of understanding anent the etiology, histo-pathology, symptomatology and treatment of cholelithiasis; (2) that the hypothesis that bacterial invasion represents the terminal rather than the primary factor in cholelithiasis has been clearly disproved; (3) that the medicinal treatment of cholelithiasis is a delusion, there being no drug which, internally administered, will cause disintegration of definitely formed choleliths; (4) that the most favorable results may be expected to accrue from cholecystostomy, cholelithotomy and temporary drainage; (5) that cholecystectomy is illogical and unwarranted excepting where the cholecyst is already damaged beyond hope of functional restoration or involved in demonstrable malignancy. SACHS.

Gall-Stone Disease—B. T. TILTON, N. Y. Med. Jour., March 4, 1916.

As far as the changes produced by stones in the gall-bladder are concerned, one finds all degrees, from a normal condition of the walls to a complete gangrene. The changes from the normal are chiefly inflammatory in character, and are dependent, not on the number or size of the stones, but on the presence or absence of infection, and particularly on the condition of the drainage of the organ. A gall-bladder may contain a hundred stones and be practically normal in size and appearance, while in another gall-bladder a solitary stone may become lodged in the entrance of the cystic duct, shut off the exit of bile, and result in a gangrenous cholecystitis, with all the inflammatory changes that this condition implies. In case inflammation arises from the presence of a stone or stones, the symptoms are fairly characteristic, particularly in the acute stage, and our efforts are exerted toward overcoming this inflammation by either medical or surgical methods. In cases, however of a stone or stones without inflammatory changes in the gall-bladder, the symptoms are often not characteristic, as long as the stones remain *in situ*. Here we have to deal with vague manifestations which often lead us to suspect other organs, most commonly the stomach. A large percentage of the cases that come to operation give a previous history, often of many years' duration, of stomach symptoms, and have been treated by physicians and stomach specialists as cases of gastritis or ulcer. Some authorities place the proportion of cases giving a previous history of digestive disturbances as high as seventy per cent. The patient complains of epigastric distress, loss of appetite, nausea, and sometimes vomiting. Vomiting is usually followed by marked relief. There are no characteristic stomach findings, but in spite of this, the patient is treated for chronic indigestion, year after year. The medical profession is not yet thoroughly alive to the fact that a large majority of these people are suffering from biliary disease. While the practitioner is waiting for his medicines to cure the dyspepsia and to alleviate the repeated attacks of pain, the patient is running the risk of developing one of the serious complications to which gall-stones give rise. The old idea is still widespread that a patient cannot be said to have gall-stones unless one or more characteristic symptoms are present. Among these may be enumerated typical gall-stone colic, pain in right shoulder blade, jaundice, enlargement of the gall-bladder, etc. Careful histories and physical examinations have shown that not more than twenty-five per cent. of those operated on for gall-stones had typical colic. As for jaundice, it is rare where the stones are confined to the gall-bladder, and is by no means the rule in stones in the common duct. SACHS.

Excretion of Certain Urinary Constituents in Hepatic Disease—*v.* MORACZEWSKI and E. HERZFELD, Zeitschr. f. klin. Medizin, Vol. LXXXII, Nos. 1 and 2.

Examination of healthy persons as well as of 4 cases of cirrhosis of the liver, 2 cases of catarrhal jaundice, one of diabetes, one of pernicious anemia, one of leukemic tumor of the liver and spleen, one of acid intoxication and 2 of chronic renal disease showed that, in general, there ensues a certain form of excretion in hepatic diseases which resembles the excretory conditions while fasting. There is a high degree of uric acid, ammonia and aceton excretion. Large amounts of volatile fatty acids and indican are always found in the urine. The aceton increase is especially noticeable when on a milk regimen. The increase of certain urinary constituents runs parallel with a decrease of others. Nitrogen, for instance, is markedly decreased. In pernicious anemia and leukemia uric acid is augmented; this, however, is not the case with ammonia, aceton and indican. In diabetes aceton and ammonia are increased; uric acid and the volatile fatty acids are not increased. In acid intoxication the volatile fatty acids, ammonia and aceton are increased, while the excretion of uric acid remains about normal. In nephritis nothing of import was noted in the excretion of the urinary constituents. Hence in diseases of the liver all aforementioned substances were increased, while in affections of the blood but a few of these substances were excreted in larger amounts.

WESTERN.

Methods of Testing the External Pancreatic Secretion—SCHLEICHER, Archiv f. Verdauungskrankheiten, Vol. XXI, No. 4.

The value of the various methods of testing the external secretion of the pancreas is still sub judice. In order to determine the reliability of the various tests author has employed the most popular methods in 22 instances in which the external pancreatic secretion seemed to be abnormal. The methods of Gross and Müller for the detection of trypsin and the diastase test of Wohlgemuth are reliable; the nuclein test of Schmidt, however, is less trustworthy. A definite opinion concerning the methods of Winternitz and Ehrmann cannot as yet be given. The oil-breakfast of Boldireff-Volhard furnishes reliable results. On the other hand, the gelodurate test of Schlecht and the glutoid capsules of Sahli gives less reliable results. The methods of Gross and Wohlgemuth may be recommended for acute cases; here the qualitative and quantitative demonstration of trypsin and diastase in the feces as well as in the urine must be made. Both tests are very reliable; they alone will evince the degree of functional pancreatic activity. Other tests may be employed to corroborate the findings obtained with the Gross and Wohlgemuth methods. WESTERN.

Morphography of Colons—J. E. DAVIS, Am. Jour. Obstet. and Dis. of Women and Children, 1916, No. 3.

Author studied the colons of 285 bodies which were embalmed with formol, phenol and glycerin compound in preparation for dissecting purposes. About 32.5 per cent. of the colons had a normal morphography. In 9.9 per cent. of all the cases, there were persisting embryologic forms. The transverse segment of the colon had descended to some point between the umbilicus and pubes in 15.5 per cent. of all the cases. Among these colons were diverticulae, gigantism, marked constructions, adhesions at the hepatic, gastric and ileocecal regions, distinct enlargements of the colons proximal to constricted splenic flexures and herniated cecums. There was a marked ptosis of the transveres colon in 23.4 per cent. of the cases and in 4.9 per cent. it was low in the pelvis. SACHS.

Intestinal Parasites in Children—H. B. WARD, Arch. Pediat., Feb., 1916.

Author concludes that intestinal parasites are more frequent than is commonly stated and that there are more varities than are usually listed in the text-books. Neither the trematodes or flukes in this country constitute a menace to the child. The pork tapeworm, tenia solium, the beef tapeworm, tenia saginata and the fish tapeworm dibothriocephalus latus though reported in all ages, even from sucklings, are of rare occurrence. Four of the cestodes reported from the human host occur chiefly in children. Davainea madagascariensis have have been reported only from tropical regions. Dipylidium caninum, a normal parasite of the dog and cat may be found in children. The nematodes or round worms furnish more parasites to children than either of the above groups. They are the ascaris lumbricoides, the stomach worm; oxyuris vermicularis, the pinworm, and trichuris trichiura, the whipworm. Hookworm disease is a disease of the young. Protozoa are extremely common in children. SACHS.

Dysentery due to Unknown Causes—v. JAKSCH, Zentralblatt f. innere Medizin, 1915, No. 47.

A case exhibiting the well-known typical clinical picture of dysentery. Bacilli or amebae could not be demonstrated. This case was undoubtedly directly produced by poisons, ptomains, after ingestion of rabbit meat. WESTERN.

NERVOUS SYSTEM

Effect of High Explosives on the Central Nervous System—F. W. MOTT, Lancet, Feb. 26, 1916.

Author states that an acquired or inborn tendency to neurosis plays an important part in the effects on the central nervous system of exposure to shell shock in a majority of cases. Of all nervous diseases, epilepsy is the one in which an inborn tendency is shown more than any other nervous affection. While shock may be a powerful exciting or aggravating cause in epilepsy, shell shock is not in itself the cause in most of the cases. SACHS.

Sensory Field of Facial Nerve—J. R. HUNT, Brain (London), Dec., 1915.

This article is a study of the symptomtology of inflammation of the geniculate ganglion. The following conditions indicate inflammation of this ganglion: (a) Pain and sensory disturbances in facial neuritis; (b) neuralgic affections; primary tic douloureux, reflex otalgia, herpetic and tabetic otalgia; (c) a sensory pathway for the direct transmission of irritative impulses to the facial nucleus, thus favoring the production of facial twitching and spasms; (d) herpedic inflammations of the geniculate ganglion, a syndrome characterized by an eruption of herpes zoster in the geniculate zone, usually associated with facial palsy and often with auditory symptoms. The cutaneous zone of the geniculate ganglion is represented on the external ear at one or more of the following places; the concha, external meatus, tragus, antitragus, incisura inter-tragica, antihelix, fossa of the antihelix superior portion of the external surface of the lobule within the auditory canal and on the tympanic membrane. SACHS.

Fracture of the Vertebrae without Cord Symptoms—P. W. ROBERTS, Surg., Gynecol. and Obstet., Feb., 1916.

Fracture of the vertebræ without cord symptoms of sufficient severity to attract attention is a condition frequently allowed to go unrecognized, yet it is not uncommon. In the past year four cases of this nature have come under author's observation and in all but one the patients were discharged from hospitals with the assurance that they were physically sound. Even though the immediate results of such an injury are apparently trifling, the broken bone may undergo a process of rarefaction and attrition with the appearance, some months later, of cord symptoms, increase of deformity, pain and weakness. The importance, therefore, of careful examination of all spine injuries is obvious. It is possible that failure to recognize lesions of this type is due to the very prevalent idea that fractures of the vertebræ are always accompanied by evidence of cord pressure. Such is the impression to be gained from most text-books on the subject which specifically state, to quote from one well known author, that in fracture of the spine "paralysis is the most important and constant symptom," while nothing is said of the possibility of fracture without this condition. In traumatism of the spine the presence of paralysis is suggestive of fracture, yet it is not conclusive, inasmuch as cord pressure from hemorrhage may produce a loss of muscular power. Of greater importance in determining the probability of fracture is examination of the spine for a point of localized tenderness. The findings will be confirmed or disproved by radiographs provided they are well taken. For this purpose apparatus of high penetration is often necessary. It is not sufficient to depend upon an X-ray plant which may be efficient for ordinary work, especially if the injury involves one of the upper seven dorsal vertebræ. In this situation the scapulæ are interposed between the plate and the tube and offer sufficient resistance to the X-rays to yield indistinct shadows unless the penetration is unusually high. The type of deformity usually produced by fractures of this nature is a long, rounded curve, differing somewhat in its appearance from the kyphos of Pott's disease which in acute cases partakes of shorter and sharper lines. SACHS.

Relation of Pelvic Diseases to Mental Disorders—A. E. PERKINS, Woman's Med. Jour., April, 1916.

(1) Pelvic disease, if causally connected with psychoses, can be regarded only as a factor entering into the causal constellation. (2) There is evidence to show that in manic-depressive insanity, pelvic disease is found more often than in dementia præcox. (3) Gynecological treatment or operation for the relief of pelvic disorders in author's cases have given satisfactory results in benefiting the mental condition in about half of the cases of the manic-depressive

group. (4) No benefit to the mental condition has been observevd after operations in cases of dementia præcox. (5) In epilepsy, in a very limited material of 3 cases, author has seen one very striking and immediate improvement in the mental state, as well as in the frequency of convulsions. In another case the result was less immediate but also quite satisfactory. (6) Gynecological operations may be one of the precipitating causes of psychoses. The severity of the operation bears no constant relation to the psychosis, and it is possible that the operation may sometimes act merely as a mental precipitating cause. (7) As to the question of operation in nervous women, not only the physical, but also the mental condition should be more thoroughly considered than is often done, and, if possible, an adjustment should be brought about before the operation is performed. (8) Sometimes the desire to be operated is a symptom of a mental condition. (9) Among 24 cases of insane imbeciles studied, 15 were known to be infected with syphilis or gonorrhea, or both. Nineteen had been sexually irregular. Nine had had illegitimate children (one 3, one 5). Two, who were married, had each 5 children. WESTERN.

Periostitis and Osteitis as a Cause of Local Pains and So-Called Neuritis and Neuralgia—C. L. DANA, Med. Rec., April 29, 1916.

The following conclusions are reached: Acute infections from the teeth, tonsils, and other foci may cause local forms of osteitis and periostitis. This fact must be reckoned with in diagnosing neuralgia and other persistent and distressing local pains. The joints are not the only region attacked by acute infection; the arthritis explanation of pains in the back and extremities has been perhaps overdone. So-called sciatica, for example, is not always arthritic or myositic or neuritic. Further study is needed to corroborate these findings and to determine the source and nature of the infections that produce them. Syphilis and tuberculosis do not play so large a part as supposed in the causation of these minor types of osteoperiostitis. Neuralgia as usually defined is a very rare disorder, and its diagnosis is to be made only after careful examination of muscles, bones, nerves, nerve centers, and sources of reflex irritations. SACHS.

Colloidal Gold Precipitating Substance in Cerebrospinal Fluid in Paresis—P. G. WESTON, Jour. Med. Research, March, 1916.

Author claims that the substance in the cerebrospinal fluid in cases of paresis which causes the precipitation of gold from colloidal suspension, is not peptone. He was unable to ascertain the exact nature of this substance. SACHS.

Infectivity of Cerebrospinal Liquid in Syphilis—R. Frühwald and A. Zaloziecki, Berliner klin. Wochenschr., Jan. 3, 1916.

There exists no regularity concerning the infectivity of cerebrospinal fluid. The liquor may exhibit spirochetes soon after infection or it may not contain them until long after this has taken place. In syphilis without nervous symptoms the infectivity will mostly ensue during the first year of the disease. It cannot as yet be stated how the infectivity of the spinal fluid is produced.

Mill.

URINARY ORGANS—MALE GENITALIA

Renal Efficiency Determination in Nephritis and Orthotic Albuminuria in Childhood—Zondek, Zeitschr. f. klin. Medizin, Vol. LXXXII, Nos. 1 and 2.

Excretion of N and NaCl occur not independently in renal affections. Release from the excretion of one constituent has also a favorable influence upon the other constituent, and vice versa. This is also the case as regards the water excretion. In orthotic albuminuria there existed only an influence upon the NaCl metabolism which was due to augmented charging with N. Western.

Chronic Retention of Urine in Children—E. Beer, Jour. A. M. A., Nov. 13, 1915.

During the past 5 years a number of children suffering from chronic vesical urinary retention have come under author's observation. Invariably the condition had been unrecognized, and the children were steadily deteriorating in health. The picture presented by these cases is very suggestive of chronic interstitial nephritis. The patients become pale and pasty. Frequently they are apathetic. Usually there is some difficulty in urination. The children complain of pain, or their parents note that they take a long time to complete the act. In some cases the straining is so great that the patient shows collapse symptoms with excessive pallor and weakness. On physical examination a globular mass is felt in the hypogastrium; it varies in size, but never disappears spontaneously. If infection is present, the mass may be tender. When a catheter is passed, this tumor vanishes. There is regularly residual urine. The walls of the bladder are hypertrophied, though this may not be evident late in the disease when excessive dilatation takes place. On palpation one may feel the bladder contract and become hard, behaving like the pregnant uterus. If no infection has set in, the urine may be clear, suggesting the urine in chronic interstitial nephritis. Eventually all the patients seem to become infected. As a secondary and constant development, the ureter openings become patulous and the ureters dilated. Unilateral or bilateral hydronephroses develop, and the enlarged kidney or kidneys become palpable. With infection setting in, the kidneys partake of the trouble, and infected hydronephrosis with or without multiple abscesses develops. Neurologic examination of these patients frequently shows nothing abnormal; in others, distinct evidence of a cord or of a brain lesion is found. Even in cases in which the most complete examination fails to reveal a definite disturbance in the nervous system, postmortem examination (3 cases) have shown definite cord lesions. Many of the cases without neurologic signs so closely simulate those with definite neurologic symptoms, as well as those adult cases of contracture of the neck of the bladder, due to some neuromuscular disturbance (arteriosclerosis of the cord, poliomyelitis, etc.), that, reasoning from analogy, one feels compelled to view these cases as due to neurologic disturbances, provided no local physical obstruction is present. In some cases a local examination may demonstrate an obstruction at the prepuce or in the urethra (stricture, folds, new growth). This should regularly be excluded, if possible. A minute meatus may be associated with a neuromuscular disturbance and, as in one of author's cases, have nothing to do with the retention. The ready passage of an instrument into the bladder probably precludes the possibility of a mechanical obstruction, whereas, in conditions of sphincter spasm, such as most of these cases probably are, no obstruction is usually felt. This corresponds to the adult cases as well as to similar spasms in other parts, notably cardiospasm. That the condition is frequently one of spasm (or perhaps better disharmony between the detrusor and sphincter muscles or relative hypertonicity of the sphincter) can frequently be demonstrated by passing a good-sized bougie into the bladder after the patient has voided. As soon as the sphincter is well stretcher and the bougie is withdrawn, the bladder may be emptied voluntarily. Again, that the detrusor is not paralyzed can be readily proved by starting the stream by forcible expression. After the sphincter spasm has been overcome the patients may void in a good stream. Röntgenographic studies of these cases with silver salts will regularly show a fair-sized bladder and dilated ureters, provided the condition has lasted a sufficient time. If the cause of the retention is a diverticulum, the Röntgen-ray cystograph will readily assist in making the diagnosis. KAUFMAN.

Observations on Pyelography—T. FAHR, Deutsche med. Wochenschr., Feb. 3, 1916.

The employment of collargol in pyelography is not followed by toxic systemic injury on account of the absorption of the drug. In case the collargol is injected under high pressure and remains for some time in the renal pelvis, finding its way into the renal structures themselves, a local necrosis of the parenchyma may ensue. For this reason great caution when injecting the collargol is imperative. MILL.

A Source of Danger in Pyelography—M. SIMMONDS, Münchener med. Wochenschr., Feb. 15, 1916.

Pyelography is wrought with a number of dangers, however the principal source of danger is not an intoxication with collargol, but a septic infection. It is absolutely necessary to ascertain whether or not the urinary bladder contains infective germs before pyelography is undertaken. If this is the case pyelography should not be made. MILL.

Metastatic Prostatitis—WILDBOLZ, Korrespondenzblatt f. Schweizer Aerzte, 1916, No. 6.

Metastatic prostatitis of non-gonorrheal origin is not often recognized. During a period of 10 years, author has seen 14 cases after influenza and 22 cases after other affections, especially intestinal disturbances and staphylococcus infections (furuncle). In 10 cases the focus could not be detected. Twenty cases were examined bacteriologically; twelve times staphylococci, 5 times associated with B. coli; twice the B. coli alone and once streptococci were found. Abscess formation took place but 5 times. The principal symptoms are frequent urination, pains in the prostatic region and during micturition, and pressure sensitiveness of the prostate. The urine is mostly entirely normal, especially in the beginning of the affection. This leads often to a wrong diagnosis. MILL.

Bacteriology of Chronic Prostatitis and Spermatocystitis—H. B. CULIVER, Jour. A. M. A., Feb. 19, 1916.

Author summarizes his article as follows: Anaerobic as well as aerobic organisms may be the exciting cause of chronic prostatitis and spermatocystitis. One-sixth of this series of cases yielded an aerobic organisms. This fact may help to explain some of the sterile cultures obtained when only aerobic media are used, as well as the inability to grow the bacteria seen on a smear. The organisms isolated on 66 per cent. of the patients tested seem to be specific for the individual infected. This is shown by the positive reactions following the injections of autogenous organisms, as compared with the negative reactions obtained with similar injections of other organisms of the same or different groups. Many instances of prostatitis and seminal vesiculitis are apparently non-infectious on a single examination, but repeated examinations often reveal the infecting bacteria. Chronic infection of the prostate and seminal vesicles seem to be partially or wholly responsible for many instances of subacute and chronic arthritis. The specific focal reactions obtained speak strongly in this direction. SACHS.

Genitourinary Tuberculosis, Diagnosis of—J. W. CHURCHMAN, Med. Rec., March 18, 1916.

Contrary to a widespread notion, renal tuberculosis may occur in a person otherwise perfectly healthy. In renal tuberculosis, renal symptoms are the exception and not the rule. In a fairly large series of cases of kidney tuberculosis which were recently seen by author—all of them advanced cases—not a single one has given symptoms on the part of the kidney. An impalpable kidney not only is no indication of the absence of renal tuberculosis, but also is no proof of the absence of an enlargement of the kidney from this disease. The sign depended upon in making the diagnosis of renal tuberculosis is the presence of tubercle bacilli in the urine. Author, however, has seen in the last year 3 cases, all proven to be tuberculosis of the kidney by microscopic examination of the tissue removed, in none of which he was able to find tubercle bacilli in the urine. This experience, however, is rather exceptional. In the vast majority of cases of renal and vesical tuberculosis not only can the organisms be found, but they can be found with great ease. In the female the diagnosis of renal tuberculosis may often be made by a palpation of the lower end of the ureter per vaginam. The cystoscope usually makes a positive diagnosis of this disease. The cystoscopic findings in bladder and kidney tuberculosis are well known. Sometimes a ureter which can readily be seen cannot be catheterized because it enters the bladder at an unusual direction. Author has seen recently a number of cases in which this difficulty was overcome in passing the right catheter into the left side, and vice versa. KAUFMAN.

The Mode of Spreading of Tuberculosis in the Male Genital System—M. SIMMONDS, Beiträge z. Klinik d. Tuberkulose, Vol. XXXIV, Nos. 1 to 3.

Primary genital tuberculosis in the male spreads in 50 per cent. of the cases from the prostatic gland, in 25 per cent. of the cases from the seminal vesicles and in another 25 per cent. from the epididymis. The infection travels testifugally as well as testipetally. Among 20 cases of testicular tuberculosis in whom castration had been performed, the autopsy did not reveal a single instance in which the operative interference had favorably influenced the tuberculous invasion of other parts of the genital system. FRY.

FEMALE ORGANS OF GENERATION—PREGNANCY—PARTURITION—INFANTS

Metastatic Carcinoma of Ovaries—W. S. STONE, Surg., Gynecol. and Obstet., April, 1916.

Carcinoma of the ovaries even though a small size occurs very frequently secondary to disease of the stomach or breast and so it is always wise to be suspicious of their metastatic nature and to be guarded in giving a prognosis. The metastasis occurs either by a direct extension through the retroperitoneal lymph nodes, by permeation or retrograde transportation, or by peritoneal inflammation. SACHS.

Diagnosis of Gonorrhea in Women—C. BUCURA, Wiener klin. Wochenschr., March 16, 1916.

The presence of gonococci can often be only demonstrated in the uterine secretion. The secretion should be obtained after disinfection of the external parts of the vagina and the lower portion of the cervix by means of alcohol or tincture of iodin. The examination should be made shortly before or after menstruation.

MILL.

Kiutsi-Malone Urinary Test for Pregnancy—F. S. CUTTER and M, MORSE, Jour. A. M. A., Feb. 19, 1916.

Authors state that there are many objections to the Kiutsi-Malone urinary test for pregnancy from the theoretical aspect. While the authors' experiments show acceptable results in 66 per cent. of the cases examined, in a remaining third, there was a high degree of uncertainty. The appearance of positive reactions in certain cases known to be negative are inexplicable, except as involving an ever-present error, such as the factors of bacteria, excreted pepsin-like enzymes, etc. The test as at present conducted cannot safely be depended on as a diagnosis of pregnancy owing to: (a) The non-specificity of the reaction; (b) the frequency of errors. SACHS.

Estimation of the Various Forms of Heart Diseases in Pregnancy—R. T. JASCHKE, Zeitschr. f. Geburtshilfe u. Gynäkologie, Vol. LXXVIII, No. 1.

Valvular disease must be rigorously differentiated from myocardial affection. It is imperative that the functional ability and the cardiac resistance be properly estimated. The clinically occurring possibilities of cardiac disease associated with pregnancy may be, (1) uncomplicated valvular disease with very little danger; (2) valvular disease together with myocardial deterioration (this combination causes most victims; (3) chronic myocardial insufficiency; (4) an acute process of the endocardium, myocardium or pericardium; (5) complications on account of extracardial disease.

MILL.

Transitory Fever of the Newborn—R. T. JASCHKE, Zeitschr. f. Geburtshilfe u. Gynäkologie, Vol. LXXVIII, No. 1.

Observations made on 1,000 newborn babies. The transitory fever is not a hunger fever. It is caused by two factors, namely, (1) the colonization of the milk flora in the intestine, and (2) the change of the colostral to the milk nutriment. MILL.

Bacterial Affections of the Urinary Organs in Infants—H. L. KOWITZ, Jahrbuch f. Kinderheilkunde, Vol. LXXXII, No. 4.

Author proposes the term "Colinephritis" instead of "Cystitis" or "Pyelitis" for the infective processes occurring in infants and which are characterized by the presence of the coli bacillus in the urine and the blood. Colinephritis affects both sexes equally often.
MILL.

Transmission of Dermatitis Exfoliativa Neonatorum—M. KAUFMANN-WOLF, Jahrbuch f. Kinderheilkunde, Vol. LXXXII, No. 4.

Report of a case. A nursing mother became locally (upon the breast) affected with dermatitis exfoliata neonatorum which was transmitted by her newborn infant.
MILL.

Acidity and Pepsin Digestion in the Stomach of the Nursling—R. KRONENBERG, Jahrbuch f. Kinderheilkunde, Vol. LXXXII, No. 5.

The degree of acidity necessary in peptic digestion of milk is smaller than in peptic digestion of coagulated ovi-albumen. The lesser degree of acidity is present in the stomach of the nursling.
MILL.

Recent Publications

THE PHYSIOLOGY OF THE AMINO ACIDS. By **FRANK P. UNDERHILL**, Ph.D., Professor of Pathological Chemistry, Yale University. New Haven, Yale University Press, 1915.

It was an excellent idea of Professor Underhill to gather in one little volume most that is known of the biochemistry of the amino acids. It goes without saying that the subject matter has been treated in an authoritative manner. Undue speculation, happily, has been avoided. The book has been divided into eleven convenient but sequential chapters, viz.: (I) The proteins and their derivatives, the amino acids; (II) digestion, and bacterial activity in relation to the amino acids; (III) the absorption of proteins and amino acids; (IV) in what form does ingested protein enter the circulation; (V) theories of protein metabolism; (VI) the further fate of the amino acids; (VII) the amino acids in relation to the specific dynamic action of proteins; (VIII) the amino acids and simpler nitrogenous compounds of foodstuffs; (IX) the specific rôle of amino acids in nutrition and growth.

The reviewer is loath to part with his copy of Professor Underhill's book. It will save him from spending many an hour over international biochemical and medical literature in obtaining data concerning the amino acids. H. S.

THEORY AND PRACTICE OF BLOODLETTING. By **HEINRICH STERN**, M.D., LL.D., Visiting Physician, St. Mark's Hospital; Consulting Physician, Methodist-Episcopal (Seney) Hospital; State Hospital at Central Islip; Deaconesses' Home; Port Chester and Glens Falls Hospitals, etc., etc., New York, Rebman Company.

This book of Dr. Stern has finally appeared in an English translation. It is the only comprehensive treatise that has appeared on this subject in English or American literature in at least fifty years. The author gives a historical retrospect of bloodletting that takes up over forty pages. The chapters on the physiology of bloodletting are especially replete with new thoughts and experimental data. It is by physico-pharmacologic action that the author explains the beneficial influence of general bloodletting upon the diseased organism. By physico-pharmacologic action he understands that influence which is exerted on the living cell by directly modifying the molecular constitution of the intracellular or extracellular fluids without synchronous alteration of the chemical structure of the protoplasm. He is of the opinion that we possess no more certain or more powerful physical remedy than bloodletting. When the indications for depletion are definite, it cannot fail, provided the amount of blood withdrawn is the proper one. "This statement," he says, "does not pertain to an eventual coarsely mechanical relief that may be afforded by the abstraction of blood, as may be the case, for instance, when the right heart is embarrassed in pneumonia. Such coarsely mechanical effect can never be counted upon with absolute certainty. However, when it is a question of a disturbed osmotic equilibrium, suspended transudation of blood plasma through the vessel walls, capillary sluggishness or arrested movement of the lymph from the tissues to the veins, then bloodletting is at once the most direct and rational remedial agent." Most of the space is devoted to the clinical part of the book. Here at great length bloodletting is dwelled upon in diseases of the respiratory organs, in circulatory disturbances, in uremia, eclampsia, narcomania, in affections of the blood, and in a variety of other diseases. Bloodletting in children is treated in a special chapter. Another chapter is devoted to the prophylactic possibilities of bloodletting.

T. F. R.

THE MORTALITY FROM CANCER THROUGHOUT THE WORLD.

By Frederick L. Hoffman, LL.D., F.S.S., F.A.S.A., Statistician, the Prudential Insurance Company of America; Chairman Committee on Statistics, American Society for the Control of Cancer; Member American Association for Cancer Research; Associate Fellow American Medical Association; Associate Member American Academy of Medicine, etc., etc. Newark, N. J., The Prudential Press, 1915.

The reviewer deplores that the space at his disposal compels him to give rather cursory consideration to the stupendous work before him. The author himself sums up the principal results of his investigation in the following words: "The actual frequency of malignant disease throughout the civilized world has been ascertained to be much more of a menace to the welfare of mankind than has generally been assumed to be the case, and that, in contrast to a marked decline in the general death rate, cancer remains one of the few diseases actually and persistently on the increase in practically all of the countries and large cities for which trustworthy data are obtainable."

The work represents an undertaking of the first magnitude. The profession is deeply indebted to Dr. Hoffman, the author, and the Prudential Insurance Company of America, the institution which so generously has defrayed the expenses arising from the compiling, printing and the free distributing of this volume of 826 pages.

H. S.

THE DESCRIPTION OF AN OPHTHALMOSCOPE. Being an English Translation of VON HELMHOLTZ'S "BESCHREIBUNG EINES AUGENSPIEGEL'S" (Berlin, 1851). By THOMAS HALL SHASTID, A.B., A.M. ,M.D., LL.B., F.A.C.S., Superior, Wisconsin, and the first translation of this classic into any language. Chicago, Cleveland Press, 1916.

This is a splendid translation of Helmholtz's epoch-making discourse upon his discovery of an ophthalmoscope for the investigation of the retina in the living eye. The translator is evidently not only an enthusiast in his field of practice, but also a man of taste, historical sense and justness. The edition is limited to five hundred copies and is destined for private distribution only. H. S.

SURGICAL OPERATIONS WITH LOCAL ANESTHESIA. By ARTHUR E. HERTZLER, A.M., M.D., Ph.D., F.A.C.S., Surgeon to the Halstead Hospital; the Swedish Hospital, Kansas City, Mo., and to the General Hospital, Kansas City, Mo., Second Edition. Surgery Publishing Company, New York, 1916.

The second edition of this book is an up-to-date exposé of local anesthesia in minor and major surgical operations. The language is clear and to the point. The illustrations leave nothing to be desired. The modern surgeon cannot afford to be without the neat little volume. S. S.

THE OBSTETRICAL QUIZ FOR NURSES. A Monograph on Obstetrics for the Graduate and the Undergraduate Nurse in the Lying-in-Room. by HILDA ELIZABETH CARLSON, New York. Rebman Company.

This is a very useful, modern compendium on obstetrics from the viewpoint of the nurse. It is a ready little reference book, which is written in very simple language. It serves its purpose very well. L. B. S.

THE
ARCHIVES OF DIAGNOSIS
Editor, Heinrich Stern, M.D., LL.D.
250 West 73d Street, New York, N. Y.
To whom all Communications of an *Editorial* Nature should be addressed

Publishers: Rebman Company
141, 143 and 145 West 36th St., New York, N. Y.

PRICE OF SUBSCRIPTION PER YEAR
(payable in advance)
To Subscribers in the United States, Canada and Mexico........... **$2.00**
To Subscribers in Other Countries (Postage prepaid)............... **$2.50**
Price of Single Copies, 75 Cents
The volume consisting of four issues comprises at least 400 pages

NOTICE TO CONTRIBUTORS

1. *The purport of the Archives is to publish papers which embody the results of original work and observation pertaining to Diagnosis and Prognosis.*
2. *Papers treating on Early Diagnosis, Functional Diagnosis, Therapeutic Diagnosis and Differential Diagnosis are especially solicited.*
3. *Contributions must not be of unreasonable length.*
4. *No contribution that has already appeared in print will be accepted for publication.*
5. *Galley Proof and Page Proof will in all instances be sent to the author for correction.*
6. *Contributors will receive 200 copies of their papers in pamphlet form free of charge. Additional copies will be furnished at cost.*

THE

ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX JULY, 1916 No. 3

FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York

PUBLISHED BY
REBMAN COMPANY
141, 143 AND 145 WEST 36TH STREET
NEW YORK, N.Y.

COPYRIGHT 1916 BY REBMAN COMPANY. ALL RIGHTS RESERVED.

Entered as Second-Class Matter, February 8, 1908, at the Post-Office at New York, N. Y.
Under the Act of Congress of March 3, 1879.

SUBSCRIPTION TWO DOLLARS A YEAR. SINGLE COPIES 75 CENTS. FOREIGN $2.50.

THE
ARCHIVES OF DIAGNOSIS
A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX JULY, 1916 No. 3

Special Articles

ON THE RECOGNITION OF THE GOUTY ETIOLOGY IN CHRONIC JOINT AFFECTIONS

(FROM THE WARDS AND THE CHEMICAL LABORATORY OF LEBANON HOSPITAL AND THE BIOCHEMICAL LABORATORY OF COLUMBIA UNIVERSITY AT THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK.)

By WILLIAM WEINBERGER
Adjunct Attending Physician, Lebanon Hospital
New York

There is no special difficulty in recognizing gout in cases where typical attacks, characteristic joint affections and tophi are encountered. There are, however, cases of atypical gout, in which the patients' complaints are vague, and the chronic multiple joint involvement closely resembles the picture of one of the many forms of "chronic rheumatism." Here the bedside diagnosis is difficult, at times even impossible. In such cases it is obviously important either to establish the gouty factor or to rule it out.

Ever since *Wollaston's* discovery of the presence of urates in tophi uric acid has been considered to be in close etiological relationship to gout. To-day's consensus of opinion overrules the dissenting voices of *Von Noorden* and *Klemperer,* who consider the impairment of uric acid formation and excretion, as they occur in gout, of a secondary nature, and assign the chief rôle in the etiology of gout to the disturbance of the purin metabolism, although admittedly not all phenomena observed in this disease can be explained on this basis.

The cleavage products of the nucleoproteins differ from those of other proteins, in that some of the substances formed are not oxidized to urea and ammonia, but give rise to the formation of purin bases—the oxypurins, xanthin and hypoxanthin, and the aminopurins, adenin and guanin—which are partly eliminated in the urine as such and partly transformed into uric acid before their elimination. These chemical processes are due to the action of various ferments, the presence of which in the organism has been established.

A part of the uric acid formed is broken up to urea in such organs as the liver, muscles and kidneys by the action of the uricolytic ferment, which so far has not been found in the blood. Uric acid may therefore be oxidized to urea, although to a slight extent; the major part will appear unchanged in the urine.

According to its origin the uric acid formed is either endogenous or exogenous; the former originating from the cell nuclei of the body itself, thus indicating a continuous destruction of the body cells—the latter being derived from the cell nuclei of the ingested food.

Considering the fact that the formation of uric acid occurs in the liver, intestinal wall, the muscles, and so forth, but not in the osseous nor the connective tissue, where gouty deposits are most frequently met with, it is obvious that uric acid must have been carried thither. Accordingly we find a distinct increase in the uric acid content of the blood of gouty individuals; and this even on a purin-free diet. Only temporarily after the intake of food containing purin substances, like thymus or liver, does the blood of healthy individuals show a similar increase in uric acid.

Of pathological conditions other than gout accompanied by an increased uric acid content of the blood, there may be mentioned the later stages of chronic nephritis with impairment of renal function, leukemia, and pneumonia during the critical stage. These conditions have to be differentiated in the diagnosis of gout.

The elimination through the urine of uric acid in both acute and chronic gout remains within approximately normal variations during the interval between attacks, although there exists an unmistakable tendency toward a lower lever (0.3-0.4 per diem), the latter especially in cases of saturnine gout.

Nor does a generous intake of fluid cause any change in the elimination of both endogenous and exogenous uric acid. (*Frank and Funk*, Deutsche med. Wochenschr., 1909, p. 519.) An attack, however, is characterized by a typical form of the uric acid elimination curve, which is so distinct as to reveal at once the presence of gout.

The gouty attack as expressed by the endogenous uric acid output in the urine is initiated by a stage of depression—the anacritical depression stage of *His*—with the development of the attack the uric acid curve rises abruptly—*Pfeifer's* uric acid flood—in order again to descend below the endogenous level—the post-critical depression stage of *Umber*.

Although this curve may be modified by the existence of cumulative attacks it remains sufficiently characteristic to be of the greatest importance in differential diagnosis.

Concerning the fate of exogenous uric acid in gout, as ascertained by the addition to the purin-free food of a definite amount of purin it is partly retained in the body, partly eliminated, though at a slower and more irregular rate than in the healthy individual, and is liable to cause a typical gouty attack. (*Bloch*, Zeitschr. f. physiol. Chem. Vol. LI, p. 472, 1907.) For the healthy organism it may therefore be said that the intake of food containing nucleins is promptly followed by an increase in the uric acid elimination.

For the gouty individual this does not hold good, at least not to the same extent. Here *Reach's* observation may be mentioned, that on addition to the food of 150 grams of pancreas the urinary uric acid was increased by 119 mgms. instead of 300 mgms., as would be expected in the healthy individual. The effect of the pancreas feeding could be seen to extend over a period of four days. This agrees with the observation of *Kaufmann and Mohr* (Deutsches Archiv. f. klin. Med. Vol. LXXIV), who ascertained the fact that the addition of thymus gland to the food of the gouty individual was followed by a more gradual ascent and descent of the uric acid curve, the summit of which was lower than in the healthy individual.

From all these facts the conclusion is justified that the formation of uric acid from nuclein is retarded in gout, or at least that the uric acid is eilminated at a much slower rate than in health—this retardation continuing for from three to five or more days, as compared to twenty-four or forty-eight hours in the normal. Mode of procedure followed in the metabolism experiments described in this paper:

The metabolism experiment is divided into (1) the fore period—lasting six days, during which the patient is kept on a purin-free, best lacto-vegetable diet—(2) the main period, which is initiated by the addition of a definite amount of purin-containing material—10 grams of sodium nucleinate (Boehringer), containing about 1.3 to 1.4 nitrogen and 2.0 to 2.5 uric acid—and (3) the after period, which theoretically begins after the effect on uric acid elimination has worn off. Practically it is best to determine the elimination of uric acid for at least four to five days after the addition of the food purin.

The daily output of uric acid in the urine as determined on the last or the last few days of the fore period represents the endogenous value. Throughout the experiment both uric acid and total daily nitrogen of the urine is ascertained. The determination of the latter is advisable, though not necessary, in order to have an approximate insight into the absorptive relations of the food purin ingested.

Theoretically the feces also ought to be analyzed, but numerous investigations in that direction established the fact that the urinary data suffice for clinical purposes.

Total nitrogen was determined by the Kjeldahl method; urinary uric acid by the Folin-Shaffer method and the uric acid content of the blood by the colorimetric method, as designed by Folin.

CASE REPORTS

The *first* case is that of Mrs. Laura S. The patient, 67 years old, was admitted to the service of Dr. Carl Goldmark at Lebanon Hospital on January 1, 1915. Father had diabetes; she had had no miscarriages; no acoholic antecedents. Complaints date back to four years ago, when painful sensations in several joints, especially the big toe of the right foot and fingers of both hands, associated with redness and swelling developed. These attacks gradually led to joint deformities.

The status exhibits a woman of poor flabby development; the heart sounds are of poor muscular quality, second aortic sound is slightly accentuated.

The joint status, taken by Dr. S. Kleinberg, January 27, 1915, reveals the following:

Under extremities normal in function and contour of joints except the last interphalangeal joints, which present a small nodule on either side of the dorsal surface of the proximal end of the last phalanx. Spine normal in contour and motion. Hips normal in contour and function. Knees are both deformed at an angle of 150 degrees with a free range of motion of about 60 degrees; both knees are enlarged, evidently through periarticular thickening; the patellae are bound down so that there is only a slight amount of lateral passive mobility. There is tenderness in the popliteal space in both sides, especially over the inner femoral condyles; on palpation of knees during motion there are felt many soft crepitations. There is slight posterior subluxation of the tibia and fibula; there is no evident fluid in knees. Feet: Double hallux valgus and depression of both metatarsal arches; outer four toes contracted; first metatarso—phalangeal joint not swollen or tender; motion in this joint produces slight soft crepitus, especially on left side.

Temperature throughout never above 100.6 deg. F.; blood count reveals slight secondary anemia; Wassermann negative; systolic blood pressure on repeated examination 150 mm. Hg., diastolic 95; urine contains faint trace of albumin and occasional granular casts; phenolsulphonephthalein excretion in two hours: 50 per cent.

Report of the X-ray department:

Left hand: General bone production with quite marked exostoses affecting especially the bones of the carpus; interarticular clefts of the last interphalangeal joints entirely missing, with anchylosis in some places; broadening of the bases of the distal phalanges and heads of the middle phalanges also quite marked cystic degeneration of the heads of the middle phalanges. Right knee: Production of bone especially around condyles of femur and tuberosities of tibia; interarticular space very much diminished. Foot: Presence of numerous moderate sized exostoses in bones forming tarsus; some anchylosis of distal phalangeal joints.

X-ray diagnosis: Chronic hypertrophic arthritis.

Clinical diagnosis: arthritis urica chronica, nephritis chronica.

EXPERIMENTAL FINDINGS:

Bedside notes state that patient's condition improved considerably during the period of purin-free diet, so that at the end of the fore period pains had almost ceased. As the record of the metabolism experiment will show, there occurred an acute exacerbation on the day the purin was added to her food.

URINARY DATA:

	Days.	Quantity cc.	Spec. grav.	React.	Total nitrogen.	Total uric-acid.	Remarks.
Jan. 30. 1915	1*	910	1016	acid	8.908	0.154	
	2	890	1016	"	9.172	0.164	
	3	865	1016	"	8.555	0.162	
	4	1085	1016	"	10.594	0.306†	10.0 SOD. NUCLEINATE
	5	1255	1014	neutr.	10.259	0.363	
	6	1270	1013	acid	10.670	0.255‡	
	7	920	1016	"	9.748	0.111	
	8	770	1016	"	8.606	0.117	
	9	970	1015	"	8.345	0.167	
	10	980	1015	"	8.035	0.196	
	11	1100	1015	"	8.659	0.221	

Chemical tests show absence of urates in tophi.

Uric acid content of blood at end of fore period (Jan. 28): 2.1 mgm. in 100 gms. of blood.

Uric acid content of blood at end of after period (Feb. 10.): 1.4 mgm. in 100 gms of blood.

Non-protein nitrogen of blood at end of fore period (Jan. 28.): 72.8 mgm. in 100 gms. blood.

Non-protein nitrogen of blood at end of after period (Feb. 10.): 88.5 mgm. in 100 gms. blood.

---

*This is actually the eighth day of purin-free diet.

†Pains in knees increased considerably.

‡Severe pains in both knees on attempt at motion; sensitiveness to deep pressure on the lateral aspects of both knees; passive motion painful and accompanied by crepitation; no swelling of either cul de sacs.

The *second* case to be reported is that of Mr. Albert S., 48 years old, who was admitted to the service of Dr. Richard Stein at Lebanon Hospital on February 16, 1915. Painter by occupation, he suffered from an attack of lead poisoning in form of a colic nine years ago. The attack lasted three months; no more attacks after the patient changed his occupation. Three years later pains developed in the small joints of both feet, especially the big toes, associated with swelling and redness. Since then had several slight attacks each winter. The last couple of years patient did not follow his trade. Of his habits it may be stated, that he partook of alcohol in the form of beer and whisky. Present attack began five days before admission with pains in the right big toe, associated with swelling and redness. Next day two toes of the left foot became involved; then the left knee, left elbow and left wrist. One day before admission small joints of left hand became involved. Status shows fairly well developed man; small tophus on helix of left ear; teeth in rather poor condition; suggestion of lead line under lower incisors.

Joint condition as described by Dr. S. Kleinberg; Slight limitation of plantar flection in both feet and hyperextension of phalangeal joints; knees and hips negative; examination of hands shows swelling and tenderness of left metacarpo-phalangeal joints of second finger; flexion painful at extreme of motion. Left elbow: Extreme flexion is painful and extension limited to 160 degrees. Spine negative. Reflexes normal.

Heart: First sound at apex of poor muscular quality; second aortic accentuated. Urine examination reveals absence of albumin, sugar, or casts, nor was lead found in the urine. Repeated examinations show systolic blood pressure to vary between 155 and 175 mm. Hg., diastolic between 120 and 130 mm. Phenolsulphonephthalein output in two hours by repeated examinations varying between 50 and 60 per cent. Weight on admission, 124 pounds, rising to 125½ at end of observation. Blood examination shows the presence of a moderate secondary anemia; no stippling of red blood cells. Wassermann negative.

Report of X-ray department:

Hands: Slight irregularities of periosteum of proximal and middle phalanges of a productive character; increased prominence of the bases of the middle and distal phalanges—exaggeration of width of bases.

X-ray diagnosis: Chronic productive arthritis.

Clinical diagnosis: Polyarthritis chronica urica—probably on a saturnine basis; arteriosclerosis.

Bedside notes reveal the frequent occurrence of pains in the small joints above mentioned, both before and during the metabolism experiment; distinct exacerbation following the addition of purin to the food.

EXPERIMENTAL FINDINGS.

URINARY DATA.

Days.	Quantity cc.	Spec. grav.	React.	Total nitrogen.	Total uric acid.	Remarks.
1*	1930	1015	acid	14.462	0.291	
2	1520	1016	"	12.348	0.291	†
3	1480	1015	"	11.986	0.264	
4	1565	1015	"	11.964	0.314	
5	1420	1016	"	11.285	0.291	
6	1380	1016	"	11.072	0.272	
7	1570	1015	"	13.071	0.286‡	10.0 Sod. nucl.
8	1025	1016	"	9.387	0.223	
9	1410	1014	"	11.384	0.218	
10	1505	1016	"	10.518	0.132	
11	1425	1016	"	10.570	0.307	
12	1470	1015	"	10.422	0.311	
13	1030	1017	"	7.952	0.211	
14	1560	1016	"	10.863	0.134	
15	1310	1016	"	10.378	0.212	
16	1390	1017	"	10.310	0.080	
17	1430	1016	"	11.365	0.218	

*This day, March 15, 1915, is the tenth day of the purin-free diet.

†Throughout the fore period patient had pains on and off in right elbow, right ankle, second right metacarpo-phalangeal joints, right terminal phalangeal joint, left tarsal and knee joints associated with moderate swelling. Toward end of fore period these pains had considerably subsided.

‡On the first day of the main period arthritic symptoms again developed; bedside notes describe swelling, heat, tenderness and exudation in left knee joint with patella floating—which symptom was not present in the fore period—left tarsal joint tender and swollen. These findings gradually disappeared, the main symptoms in the after period being stiffness in the joints previously affected.

Examination of tophus shows absence of urates.

Uric acid content of blood during fore period (March 11, 1915.): 0.93 mgm. in 100 gms. of blood.

Uric acid content of blood at end of after period (March 31, 1915.) 2.5 mgm. in 100 gms. of blood.

Third case: Mrs. Anna Sch., 43 years of age, a private patient of Dr. S. Basch at Lebanon Hospital, has been married 23 years, has six children, all of whom are living and well; there were two miscarriages—the fourth and last pregnancy. Her complaints were rather vague, beginning in the winter of 1913 with swelling of and pains in the second phalangeal articulations of right hand. In the course of the following six to eight months the small joints of the left hand and foot became painful; also pains in both shoulders, neck and occiput.

At the time of admission, February 11, 1915, her complaints were confined to pains in the neck, especially on awakening in the morning; the pains in the fingers were of less intensity than before, a sensation of stiffness remaining; no more pains in knees or feet.

Status: Patient rather underweighed (101 pounds); teeth in poor condition; internal organs apparently normal; urine analysis reveals the presence of a very faint trace of albumin, but no casts; sugar absent. Phenolsulphonephthalein output: 60 per cent. in two hours. Systolic blood pressure: 125, diastolic: 95 mm. Hg. Slight secondary anemia; blood culture negative; Wassermann negative; weight during stay in hospital varying between 101½ and 103 pounds.

Orthopedic examination by Dr. S. Kleinberg (February 13, 1915):

Right shoulder is free in motion in all directions except outward rotation, which is limited only at extreme of motion; there is a distinct atrophy of the infraspinatus and teres minor muscles suggesting an old subdeltoid bursitis. Left shoulder and both elbows are normal in contour and function. Both hands show hyperextension (slight) of the terminal interphalangeal joints of the second to fifth fingers and flexion deformity of the first interphalangeal joints of the fifth fingers; the proximal interphalangeal joints of the fifth fingers are not swollen and are free in motion except the extreme of extension, which appears limited by contraction of the soft tissue on the palmar aspect. Both ankles are free in motion; feet show double hallux valgus, contracted toes and corns, all evidences of patient having worn conventional shoes. There is fullness behind left external malleolus along the site of the peroneal tendons; there is no other swelling about the ankles or small joints of either foot. Hips and knees normal.

Report of X-ray department:

Examination of hands shows presence of quite marked bone absorption.

X-ray diagnosis: Chronic atrophic arthritis.

Clinical diagnosis: Mild form of chronic arthritis deformans.

Bedside notes prove that no change occurred throughout the metabolism experiment either in objective findings or subjective complaints.

EXPERIMENTAL FINDINGS.

URINARY DATA.

Days.	Quantity cc.	Spec. grav.	React.	Total nitrogen.	Total uric acid.	Remarks.
1*	735	1022	acid	7.677	0.306	
2	790	1022	"	9.029	0.494	10.0 Sod. nucleinate
3	925	1020	"	9.638	0.380	
4	1025	1020	amph.	9.207	0.324	
5	940	1019	acid	8.751	0.312	
6	875	1023	"	9.493	0.338	
7	660	1026	"	8.592	0.328	

*This is the eighth day of purin-free diet. (February 20, 1915.)

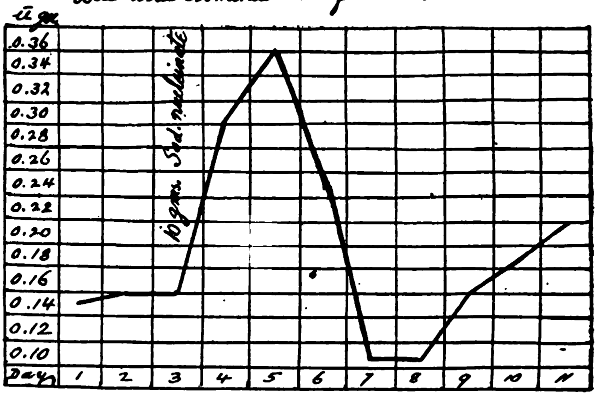
Uric acid content of blood taken at end of fore period (Febr. 19, 1915) = less than 0.5 mgm. in 100 gms. of blood.

DISCUSSION OF RESULTS

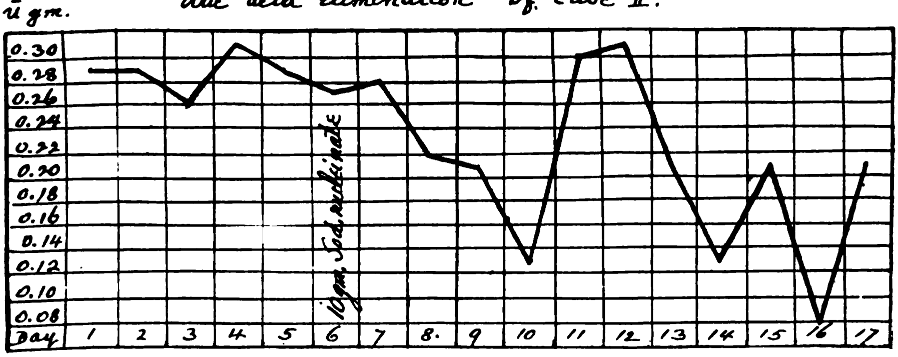
1. Uric acid content of the blood.

It is an established fact, that gouty blood contains larger amounts of uric acid than normal blood, and this even on a diet free of purin. *Folin and Denis* (Jour. Biol. Chem. Vol. XIV., 1913) believe, that from 1 to 2 milligrams of uric acid in 100 grams of blood represent the normal variations; these authors found 5.5 milligrams in "uric acid blood." It is also well known, that a temporary increase in the uric acid content occurs in healthy individuals after the ingestion of food rich in purin substances like thymus, liver, and so forth. A perceptible uricemia also occurs in other pathological con-

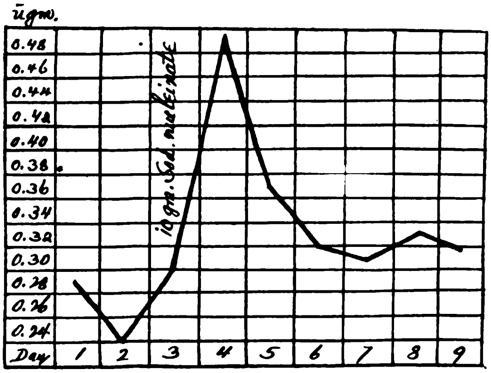
Uric acid elimination - of case I.



Uric acid elimination of case II.



uric acid elimination of case III.



ditions like leukemia, the critical stage of pneumonia and the retention stage of chronic nephritis, where the elimination of nitrogenous waste products such as urea, non-protein nitrogen and also uric acid is interfered with. Prolonged Röntgen treatment is apt to produce an artificial uricemia, which is caused by the destruction of enormous numbers of leukocytes. It is obvious, that all these conditions will have to be differentiated against in the attempt to make the diagnosis "gout" from the increased uric acid content of the blood alone. It may be mentioned that reliable methods are subtle and at the present time rather complicated, as is proven by the many attempts at simplification.

Case I with a "blood uric acid" of 2.1 and 1.4 milligrams in 100 grams of blood on a purin-free diet exhibits a uricemia beyond the normal. Considering the high non-protein nitrogen content of the blood—72.2 and 88.5 milligrams in 100 grams of blood—the question arises, whether the presence of a nephritis may not be the cause of this high uricemia, especially as the urinary findings point in the same direction. However the phthalein output of fifty per cent. in two hours points to a fair functional capacity of the kidneys, this furnishing evidence somewhat conflicting with the high rest nitrogen content of the blood.

In case II the uric acid content of the blood is 0.93 and 2.5 milligrams, respectively, in 100 grams of blood on a purin-free diet. This uricemia must also be considered abnormally high. Although neither clinical nor laboratory findings point to a complicatng nephritis, it was still considered probable by clinical observers at the bedside, that the kidneys may have been to a certain extent involved in the general arteriosclerotic process exhibited by the patient.

Case III shows a uric acid content of less than 0.5 milligram in 100 grams of blood.

Renal function was not found deficient, both urinary findings, blood pressure and phthalein output being normal.

It would apparently be unwise to make or exclude the positive diagnosis of gout from the above findings.

Cases I and II may possibly exhibit symptoms, that are due to both gout and nephritis, especially as the etiological interrelation of these two conditions is a matter of frequent observation.

2. Presence of tophi and their examination for urates.

The presence of undoubted tophi is of course pathognomonic of gout. Frequently, however, doubt arises as to their absolute identification, as there is a possibility of other formations like small fibroid nodules on the margin of the ear, and especially small sebaceous cysts closely simulating tophi.

A microscopical and chemical examination is supposed to bring information as to their identity; the finding of biurate crystals with the absence of oil droplets and epithelial cells in the contents furnishing the means of differentiation.

The records of cases I and II show that the presence of urates in the tophi could not be ascertained even by the murexid test. This is in accord with the findings of *M. Kahn* (Arch. Int. Med., Jan., 1913, Vol. XI., pp. 91-99), who may be quoted as saying in this connection: "Gouty tophi are not always composed of uric acid or the urates. In three concretions examined a negative murexid test was invariably obtained."

It may be said therefore, that, where urates are found in the formations, that impress the observer as being tophal in character, the diagnosis of gout is established, while a negative test does not exclude it.

3. Relatively low endogenous urid acid output.

If we scan over the records of our cases, we find the average endogenous uric acid value in case I 0.160, in case II 0.287, in case III 0.306.

These rather low figures would speak in favor of the diagnosis of gout in all three cases. However, in the light of subsequent results the fallacy of this argument becomes apparent. It is this lack of decided differences, which renders the consideration of this factor valueless.

4. Delayed elimination of the exogenous uric acid derived from an addition to the food of a definite amount of purin material.

This delay is conspicuous in case I, where the elimination of 0.444 milligram of uric acid in excess of the endogenous value extended over a period of three days.

Case II is interesting in that the purin addition apparently did not give rise to an increased output of uric acid at all, but led to a retention, which of course is tantamount to an extreme delay in elimination.

Prompt was the response to the purin addition in case III, the exogenous uric acid being eliminated in two days.

According to these findings cases I and II, with an apparently delayed exogenous uric acid elimination, have to be considered apart from case III, which shows no impairment of its purin metabolism. From this point of view cases I and II would apparently answer the requirements laid down as indicative of gout, provided any other factor like a complicating nephritis could be excluded. In the first case the presence of a nephritis was established beyond doubt; in the second the possibility of a renal insufficiency has to be considered.

Accordingly there is still some doubt as to the etiology of cases I and II. But recent investigation rendered the reliability of the method of differentiating joint affections from the sole ascertainment of a delayed exogenous uric acid elimination still more doubtful. *M. Ljungdahl* (Zeitschr. f. klin. Medicin, Vol. LXXIX., 1914, pp. 177-200), who utilized this method to distinguish gouty from non-gouty forms of polyarthritis, came to the conclusion, that, while the elimination time of exogenous uric acid is undoubtedly shorter in non-gouty cases as compared to cases of gouty character, this difference neither occurs in all cases promptly nor is it pronounced enough *per se* to decide the diagnosis.

5. Observation of the characteristic uric acid curve during an attack.

In considering the elimination curves of the three cases studied let us first analyze that of case III. The addition of ten grams of sodium nucleinate on the third day of the experiment was promptly followed by a rise in the uric acid elimination, that lasted two days. The decline following this rise halted at the normal endogenous level, which was then maintained. We see herein the expression of a normal purin metabolism.

Curve II makes an altogether different impression. The purin diet causes the uric acid value to go far below the average endogenous value—from 0.28 to 0.13—then the curve rises to attain its highest point—0.31—six days after the purin was taken, in order to descend again to a very low level—0.08—after which an unmistakable tendency to attain the endogenous level can be observed.

This behavior of the uric acid elimination is characteristic of a gouty attack; the anacritical depression, the uric acid flood—in this case of a relative character—and the postcritical depression; all being easily recognizable in the curve. In accord therewith are the clinical symptoms exhibited by the patient during this experiment, consisting in acute joint pains, that were intensified by the intake of the food purin.

Of similar import is curve I, which distinctly shows the uric acid flood and the postcritical depression stage, although the anacritical depression is missing—a fact of frequent observation. Here also the patient shows an exacerbation of his joint symptoms following the intake of the food purin. The existence of gout in this case may therefore be considered established.

CONCLUSION.—Of the laboratory findings pointing to the gouty etiology in chronic joint affections the most reliance may be laid on the presence of undoubted tophi and on the observation of the characteristic uric acid curve during an attack. The attack may occur spontaneously or may be produced by the addition to the food of a definite amount of purin. Although it has not been shown, that the ingestion of purin gives rise to an attack in all cases of polyarthritis due to gout, in the cases presented the attack developed promptly.

While the diagnosis of gout may be further supported by other concurring data like high uricemia, low endogenous uric acid output, and delayed elimination of exogenous uric acid, these factors alone are either ambiguous or not distinct enough to be of any decisive value.

The writer wishes to acknowledge his indebtedness to the medical board of Lebanon Hospital for permission given to carry out this work and takes great pleasure in expressing his thanks to Dr. Gies for the interest taken and laboratory facilities extended.

EARLY DIAGNOSIS OF ACUTE INFANTILE PARALYSIS

By LOUIS C. AGER
Chief of Staff, Kingston Avenue Hospital, Poliomyelitis Service
Brooklyn-New York

To describe under this old title a series of symptoms which does not include paralysis is incongruous but it is doubtful if the various other names which have been suggested for this particular infection will ever supplant the term which has become so firmly fixed in medical literature. In this brief paper no space will be devoted to the discussion of the typical resulting paralyses of the disease, but an attempt will be made to present the other symptoms upon which a tentative diagnosis may be based, subject to confirmation by spinal puncture. The symptoms fall into two greups.

First: Those which indicate an acute general infection of some kind—premonitory symptoms.

Second: The special symptoms which indicate the particular nature of the infection, diagnostic symptoms.

The premonitory symptoms vary in extent and severity as widely as do the early symptoms of scarlet fever. Fever, malaise, vomiting, headache, convulsions, pharyngeal and tonsilar irritation, constipation with abdominal distress, more rarely diarrhea may or may not be present in varying degrees, indicating that an acute infection of some kind has occurred. In time of epidemic such symptoms should be regarded with suspicion, but they have no pathognomonic significance. The stress laid upon the infectious nature of the discharges from the mouth, nose and throat has given an erroneous impression to the laity and, to a certain extent, to members of the medical profession, that catarrhal symptoms are frequent. In my experience, increased secretions from the mouth, nose and eyes is practically never present except in the later stages of severe infection.

Only cases in which the illness shows no other symptoms should be called abortive—that is, this term should be restricted to those cases in which there is no evidence of infection of the central nervous system. We have no method at present of accurately recognizing such cases. It is reasonable to hope that some specific reaction analogous to the von Pirquet and Schick tests may be devised as a result of laboratory investigations. It is very generally assumed, however, that the number of such cases is large and that they are the disseminators of the contagion. I am inclined to believe that further investigation will show that a very large majority of individuals cease to be infectious or "open cases" very early in the disease.

There is still another class of cases seen in large numbers during an epidemic, most of which ought to be recognized, but are not on account of the skepticism of private physicians and Public Health officials. I refer to the patients who show definite evidence of the invasion of the central nervous system by the organism, without any appreciable resulting paralysis. I hope that as a result of the present epidemic these will be more generally recognized at all times, and that the tentative diagnosis will be proved or disproved by careful examination of the spinal fluid.

The early diagnostic symptoms, of course, show great variation in intensity and duration, and they may, therefore, escape recognition unless a thorough physical examination is made early in the acute stage of the disease. They are not pathognomonic in a strict sense, as they may be the result of other infection of the central nervous system. They may be divided into symptoms of irritation of the psychic, the motor and the sensory tracts.

Psychic inhibition is quite characteristic of the disease, and may vary from mere drowsiness to coma. The coma, however, varies from that of meningitis in that the patient is easily aroused to almost normal mentality, and the special senses respond readily to direct stimulation. On the other hand, some children show marked excitability and a peculiar suspicious watchfulness of what goes on about them. In severe infections the mental excitation may go on to a state of delirium resembling that of typhoid fever. I have only seen a few such cases, but they have also been noted by Wickman and others.

Many of the early symptoms observed in the skeletal muscles are conscious or reflex attempts to prevent pain and are not due to invasion of the motor tracts of the cord. The most frequent symptom of this class is anterior-posterior cervical stiffness, which is usually accompanied by a similar rigidity of the spine. The patient may also show a tendency to resist movement in any or all of the extremities. I have seen one case in which scorbutus was mistaken for poliomyelitis, and similar instances have been recorded by others. Later in the disease, particularly in the meningitic type, we may see true spastic rigidity, scaphoid abdomen, and convulsions, just as we do in other forms of meningitis. Increased knee jerk and ankle clonus are occasionally noted. We may find that they are common early transitory symptoms of considerable diagnostic value, followed later by the loss of reflexes so frequently noted. Fibrillary twitching of the affected muscle has been mentioned as a late symptom, but I have also seen it before any definite signs of paralysis.

Sensory symptoms apparently vary in frequency in different epidemics. In the present epidemic they seem to be present in practically every case. It may be that the more careful medical attention that the patients are now receiving is responsible for this apparent change in the nature of the disease. Spontaneous pain and varying degrees of hyperesthesia are often noted in the so-called polyneuritic type—better named pseudo-neuritic. The pain and tenderness along the nerve trunk is very marked and usually requires the free use of codeine or morphine. The skin and muscles of the back almost invariably show some degree of sensitiveness and spontaneous pain early in the disease, soon followed by muscular pain and tenderness in the extremities. This is the reason that so many of these patients are treated for rheumatism. The skin tenderness may be so severe as to make the weight of the bedclothes unendurable.

In addition to these three groups of symptoms, there are frequently indications of disturbances of special centers, such as those which control sweating, local and general temperature, and circulation. Marked sweating, even with high temperature, is a frequent early symptom and I have seen later as much as a degree and a half sub-normal rectal temperature in a mild case. The lower temperature of the paralyzed limb is a well recognized condition, but this change in temperature is occasionally observed early in the disease. Disturbance of the circulation may be indicated by a tache, by irregular lividity or mottling, or by marked localized flushing.

If the symptoms briefly noted in this paper are sought in all suspected cases, a much larger percentage of those infected with the poliomyelitis organism will be recognized early enough in the disease to receive helpful treatment, and if some mistakes in diagnosis are made, they may be corrected by spinal puncture.

ECHINOCOCCUS CYST OF THE LEFT LOBE OF THE LIVER DISCHARGING INTO THE LEFT HEPATIC DUCT

By RUSSELL S. FOWLER
Chief Surgeon, First Division, German Hospital of Brooklyn; Surgeon, Methodist Episcopal (Seney) Hospital; Consulting Surgeon, Caledonian Hospital and Hebrew Orphan Asylum
Brooklyn-New York

Cases of echinococcus cyst involving the biliary passages upon which operation has been done are very rare. Cases have been reported by Duval, Langenbuch, Sasse, Le Jeuntel (Alençon), Borchard (Paen), Page, Rossi, von Huber, v. Mosetig-Moorhof, Körte and Kehr.

Rupture of such cysts into the biliary passages is very uncommon. Rupture causes sudden blockage of the common duct and is the occasion of very severe biliary colic. There is profound collapse and death has been known to immediately follow. Frequently the blockage is diagnosed as calculus obstruction. A correct diagnosis is only possible if echinococcus elements are found in the stool. In some cases there have been reported recurring colicky attacks and intermittent common duct obstruction; this occurs when the communication between the cyst and bile passages is small and closes from time to time. In other reported cases there were no colicky attacks but symptoms of cholangitis. One fatal case presented the appearance of acute yellow atrophy of the liver. In cases in which the cyst does not present as a tumor the differential diagnosis is between liver abscess, cholangitis with chronic stone or tumor blockage of the common duct; the diagnosis is only possible when cyst elements are recognized in the feces. Occasionally rupture occurs without symptoms; then also the condition can only be recognized by the appearance of echinococcic elements in the stools. In some cases there has been rupture into the veins of the liver or inferior vena cava followed by the carrying of embolic cyst elements to the heart and pulmonary arteries occasioning immediate death. The usual symptoms of rupture of an echinococcic cyst into the biliary passages are severe pain, chills and fever. The obstruction in the common duct accompanying the rupture may be by impaction of cyst contents or by inflammatory swelling caused by the intense irritation.

The case herewith presented gave the following history: M. R., female, age 21, married, No. 33205, seen in consultation with Dr. Calogero Giovinco Nov. 12, 1915. About four months before the patient had noticed that her urine was high colored and she became jaundiced; after three days this jaundice disappeared. Two weeks ago she had pain in the epigastrium, diarrhea and vomiting. This pain continued and she vomited from time to time. Examination showed the abdomen extremely tender below the right costal border and over the right lobe of the liver. There was rigidity of the right rectus muscle. Moderate jaundice.

Immediate operation was done. The liver was found enlarged and acutely congested; the edges rounded. Intense inflammation of the gall-bladder and ducts with all the glands along the cystic, hepatic and common ducts acutely enlarged. Locally intense peritonitis. Gall-bladder opened and found packed with fine brilliant yellow sand. Walls thickened and acutely inflamed. Gall-bladder cleansed. Cholecystostomy. Two split tube drains. It was not thought wise to explore the ducts on account of the extreme and unusual acute inflammation. It was thought in all probability the same yellow sand would be found throughout the ducts and that it would be impossible to cleanse the ducts with the existing inflammation.

After course: The jaundice slowly subsided but never entirely disappeared, a little yellow tinge persisting in the eyes. Pain subsided. Bile flowed from the tube and appeared in the feces. It was deemed advisable to continue gall-bladder drainage for a considerable period but unfortunately the tube became displaced on the tenth day and this fact was not discovered until the day following. An attempt to replace the tube was unsuccessful. The patient continued free from pain except slight epigastric cramps from time to time; gradually gained strength though a slight degree of jaundice persisted. The wound healed slowly. There was but slight discharge of bile from the wound.

12. 8. 15. Discharged. Wound healed. Very slight jaundice.

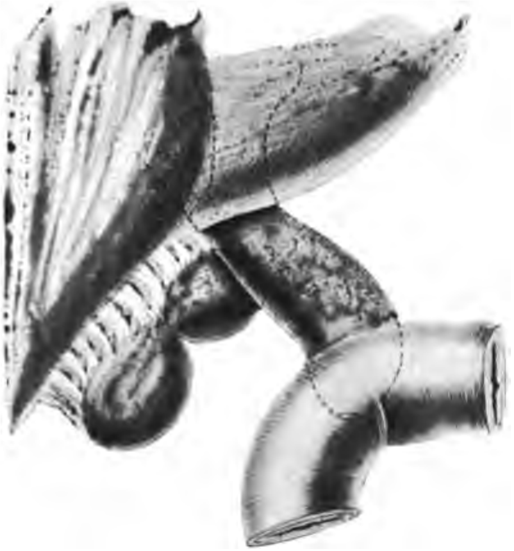
12. 22. 15. Severe epigastric cramps and slight increase in jaundice.

12. 26. 15. More severe cramps and pain over the gall-bladder and liver. Readmitted to hospital. Slight jaundice. Liver enlarged, two fingers breadth below free border of ribs. Gall-bladder region sensitive. Wound firmly healed. Operation was delayed on account

of the difficulty which it was felt would be experienced in cleansing the ducts. However, pain increased and jaundice deepened slightly so that operation could no longer be delayed.

Operation: 1. 6. 16. Incision through previous scar. Many dense adhesions. Gall-bladder adherent to scar; acutely distended; opened. Contents, thin bile; mucous membrane intensely congested; hour glass type of gall-bladder. Colon, omentum, duodenum, liver and stomach densely adherent to gall-bladder and neighborhood. Adhesions separated carefully and normal anatomical relationship of parts restored. Cystic duct dilated to twice normal. Common duct dilated to the size of the duodenum or even larger. Bile aspirated from common duct by suction apparatus. Duct incised, walls two to three times normal thickness; escape of bile and bile stained membranous detritus. This membranous detritus varied in size from pin point to the size of the last joint of the ring finger. Metal catheter introduced into common duct and duct washed out with saline, the opening into the duct having been enlarged sufficiently to permit of the ready escape of the saline alongside the catheter. Over a pint of detritus, stringy, glutinous, stained black with bile, was washed out in this manner or removed by a long curved blunt curet introduced for several inches, ten inches at times, into what was taken to be the left hepatic duct. In the detritus were numerous pieces of thick green membrane, recognizable as probable echinococcus cyst lining. Two gallons of saline were used. A large probe passed freely into the duodenum. Some saline, about a pint in all, was allowed to run into the duodenum from time to time. When the saline from the duct returned clear the curet was passed up and fresh membrane removed and the flow again started. This was repeated until so far as could be ascertained the ducts were clean. The curet passed ten inches into the left hepatic duct without meeting resistance. There was no bleeding from the irrigation. Digital exploration revealed a large opening of the common bile duct into the duodenum, an opening large enough to admit the tip of the finger into the duodenum; the common duct itself was enormous, the finger was lost in it; toward the liver the finger felt an opening about twice the normal size, taken to be the right hepatic duct opening and a continuation of the common duct taken to be the left hepatic duct, which freely admitted the finger. The latter was fully as large as the enlarged common duct, the size of the duodenum (see illustration). The cystic duct opening could be demonstrated and was twice the normal size. The lining of the common duct and the left hepatic duct was smooth. The lining of the common duct so far as could be seen presented no evidence of inflammation. The patient was thin, worn out by long illness and bore the operation very badly. The pulse, while the beats were distinct, was extremely rapid, rising to over 200 per minute during the irrigation of the hepatic duct. It was felt, however, that the patient stood very little chance of recovery unless this duct could be freed entirely of its impacted detritus. When the ducts were apparently clean and the irrigation discontinued, the pulse fell to 180, but it was of very poor quality. Continuous hypodermic whiskey stimulation was given from the time the pulse became weak and saline and whiskey were administered by rectum. A rubber tube, with a fenestrum cut in the side, was placed in the left hepatic duct through the common duct incision in such a manner as to place the fenestrum facing toward the duodenum. This was used to take the place of a T tube which was not available. A large caliber tube was used not only for the purpose of freer drainage and to avoid blockage but also with the idea of allowing of irrigation of the hepatic duct and common duct should blockage occur, or should some of the detritus show in the discharge from the tube. The incision in the duct was sutured around and to the tube. A split tube was placed in the foramen of Winslow; a second split tube in relation to the junction of the cystic duct with the common duct (which juncture was higher than usual); a rubber tube was placed in the gall-bladder and the gall-bladder edge inverted. The condition of the patient did not allow of cholecystectomy although otherwise this was indicated. A rapid layer closure of the abdomen was effected. The patient left the table with a pulse of 180, very weak.

After course: General treatment of shock (heat, posture and saline by rectum) in addition to direct cardiac stimulation with half strength whiskey by hypodermic, continuous at first, then every two minutes, later every five minutes and so gradually stopped, succeeded in accomplishing restoration to a fairly good condition in twelve hours; at the end of twenty-four hours the bed was leveled and the patient out of danger so far as the operative procedure was



ECHINOCOCCUS CYST OF THE LEFT LOBE OF THE LIVER DISCHARGING INTO THE LEFT HEPATIC DUCT

Russell S. Fowler

concerned. The jaundice was less marked. At no time had it been severe. For the first forty-eight hours the discharge through the duct tube connected with a subaqueous drain was free and consisted of clear bile.

1. 8. 16. Detritus began to pass through the duct tube and it was feared that blockage might take place. Eight ounces of saline were slowly introduced into the common duct, the subaqueous drain being also disconnected and cleaned. The introduction of saline produced no pain. The patient noticed a sensation of warmth in the epigastrium. There was a return of about one ounce of saline through the gall-bladder tube. The return flow through the duct tube contained mucous and thick stringy material, blood streaked. About two ounces of the saline was not returned. Following this flushing of the common and hepatic ducts the discharge from the duct tube again became free and clear bile. As much as ten ounces of bile was collected in the bottle in twelve hours.

1. 9. 16. The flushing was repeated, there having appeared some detritus in the discharge. This detritus consisted of echinococcus cyst lining. At this irrigation seven ounces of saline was used, two ounces at a time being slowly introduced and then a wait of a few minutes. The return flow again contained mucous and thick stringy reddish material. About two ounces of saline returned through the gall-bladder tube. Practically all of the saline returned. The patient complained of slight pain during the irrigation.

1. 10. 16. The common duct was irrigated, seven ounces of saline being used. Return flow as before; two ounces were returned through the gall-bladder tube. Practically all the saline returned.

1. 11. 16. Common duct irrigated; return flow about the same; some saline, about two ounces, returned through the gall-bladder tube. Patient noticed scarcely any pain. A small amount of saline escaped through the wound alongside the tubes.

1. 12. 16. Return flow the same. Saline returned through the gall-bladder tube and a considerable amount escaped through the wound alongside the tubes.

1. 13. 16. Sutures removed; good wound union except at emergence of tubes. Split tube removed.

From this time on the patient was comfortable. Free biliary drainage continued from the gall-bladder tube and from the duct tube. The tubes were removed on the seventeenth day. Further convalescence uneventful. The wound discharged a good quality of bile and the stools became normal.

2. 5. 16. Discharged. Wound reduced to a small sinus. No biliary discharge. Patient strong.

Pathological Examination: The detritus removed at operation showed everywhere echinococcus cyst wall and hooklets. No daughter cysts could be demonstrated. Examination of the detritus passed through the common duct tube post operatively showed characteristic hooklets. Repeated examination of the stools failed to show echinococcus elements.

2. 23. 16. Wound firmly healed. Patient has remained free from symptoms and is rapidly regaining her strength.

5. 21. 16. Patient has remained well.

CHOLECYSTITIS CATARRHALIS SUBACUTA; AN EARLY TYPE OF GALL-BLADDER DISEASE

By RUSSELL S. FOWLER

Chief Surgeon, First Division, German Hospital of Brooklyn; Surgeon, Methodist Episcopal (Seney) Hospital; Consulting Surgeon, Caledonian Hospital and Hebrew Orphan Asylum

Brooklyn-New York

The time has passed when a patient the victim of gall-bladder disease goes through life, suffering at times untold agonies or at the least a sufferer from "indigestion" and restricted as to diet and pleasures.

The operation of cholecystectomy done by expert hands in the early stage of gall-bladder disease will obviate all discomfort and restriction as to mode of life in those early sufferers from the disease who are fortunate enough to consult wise and far seeing physicians.

The following *outline* is the result of a careful study of a number of cases seen over a period of twenty years. But two illustrative cases are cited out of perhaps a dozen observed of what is a very early stage in gall-bladder disease.

Clinical type of disease:

Simple gall-bladder colic.

Pathology (Cholecystitis catarrhalis subacuta):

No adhesions.

No marked gross evidence of past or present inflammation (villi are more prominent than usual).

Cystic duct patent.

Contents of gall-bladder: No stones or single or multiple stones usually very small; clear or slightly turbid bile, in most instances sterile.

Microscopic:

Mucosa infiltration with lymphocytes and leukocytes.

Appearance of gall-bladder in situ:

There is absolutely no evidence of pathological change. The gall-bladder is not abnormally distended. Careful palpation *may* reveal small stones but is not to be relied upon. The diagnosis can only be verified by opening the gall-bladder and then only at times by the eye of one versed in the pathology of the condition. Enlargement of the gland of the cystic duct at times seen.

Symptoms:

An occasional brief colicky cramp in the epigastrium sometimes accompanied by tenderness over the gall-bladder (due to temporary blocking of the neck of the gall-bladder) and at times radiating around to the back. (If small stone is passing ducts more severe pain and in some instances temporary jaundice.)

Physical examination:

Negative. During attack tenderness over the gall-bladder can at times be elicited on palpation. (If small stone is passing ducts perhaps temporary jaundice.)

Differential diagnosis:

Right movable kidney.

Epigastric hernia.

Peptic ulcer.

Chronic appendicitis and other lesions causing intermittent irritability of the pyloric sphincter as their primary symptom but which have not produced a sufficient pathological change in themselves locally to allow of a tentative diagnosis, i.e., kink or partial obstructions in the course of the small and large intestines with their manifold pathology (angulation, dilatation, stenosis, lateral twisting), Meckel's diverticulum. Simple indigestion.

Treatment:

If attacks are frequent (every few months) whether disabling or not, operation. If at operation all stones can be readily removed without trauma and there is absolutely no gross change in the mucosa, *cholecystostomy;* otherwise *cholecystectomy.* My own preference is for cholecystectomy, for these cases seem to me to be the initial stage in gall-bladder disease. Whether the slight changes in the mucous membrane precede, accompany or follow the formation of stones it is impossible to say. Occasionally with the same symptoms and same pathology no stones are found. This would seem to indicate that the pathologic changes precede the stone formation, *but* it might easily be true that the very small stones had passed.

As illustrative of this pathological type I cite the following two histories:

Serial No. 331. M. E. H. No. 29195. N. D., aged 30. Manager, East Orange, N. J. Referred by: Dr. Thomas C. Craig of Brooklyn. Admitted: 8. 25. 07. Operation: 8. 26. 07, Cholecystectomy. Discharged: 9. 13. 07. Cured.

History: Typhoid fever ten years before. Eight months ago severe abdominal cramps and backache accompanied by nausea. Six similar attacks in past six months. Abdominal pain not localized but extended up into chest. Attacks lasted from 2 to 24 hours. The last few attacks have been more severe than the previous ones and required chloroform. Patient would at times become delirious from pain. During and for a time after the attacks the stools were clay-colored and the urine very dark. Evanescent, slight jaundice. Last attack one week ago. Following this attack slight tenderness persisted in the region of the gall-bladder.

Examination: 8. 20. 07. Negative except slight tenderness in region of gall-bladder and moderate bile in urine; very slight jaundice.

Diagnosis: Cholecystitis, catarrhal, subacute.

Operation: 6. 26. 07. Gall-bladder apparently normal; no palpable stones; ducts normal; no glandular enlargement. Gall-bladder opened. Mucous membrane hardly noticeably changed; yellow bile in bladder becoming slightly thicker and turbid in lower part of gall-bladder; a very few small soft brilliant yellow "stones" slightly ovoid in shape like minute rolled up masses of yellow clay. Cholecystectomy from below upward; drainage of cystic duct.

Pathological examination: No gross change in gall-bladder except in mucous membrane, which was slightly thickened and injected. Culture negative.

After course: Uneventful. Sinus closed in three weeks.

Subsequent history: Well and has been free from symptoms, 9. 30. 14.

Comment: This case I believe to be illustrative of gall-stones in the process of early formation. The little eliptical rolled up masses of biliary salts would, no doubt, if left in situ have developed into full fledged stones.

Serial No. 493. E. N. aged 23. Housewife. Brooklyn. M. E. H. No. 56554. Referred by: Dr. Heinrich Stern of New York. Admitted: 11. 29. 15. Operation: 11. 30. 15. Cholecystectomy. Discharged: 12. 19. 15.

History: Repeated sudden, sharp, disabling attacks of epigastric pain which radiated around the right costal margin to the back and were accompanied by jaundice. First attack 9. 1. 15. lasted 24 hours, accompanied by jaundice. Second attack one month later of a similar nature but with the added symptoms of chills, nausea and vomiting; lasted three or four days. Third attack 11. 15. 15. of same character as second attack. No symptoms between attacks except occasional discomfort in the right iliac fossa not related to the other pains.

Examination: 11. 20. 15. Slight tenderness in gall-bladder region and in right iliac fossa at the usual site of the appendix.

Diagnosis: Cholecystitis, catarrhal. Appendicitis, chronic.

Operation: 11. 30. 15. Liver normal. Gall-bladder appeared normal, about ¾ distended. No evidence of inflammation. No adhesions. Careful palpation revealed numerous very small stones in the gall-bladder, one very small stone in the extreme upper portion of the cystic duct (free and readily propelled upward into the gall-bladder). No stones in the other ducts. Gall-bladder aspirated with vacuum apparatus.

No thickening of the ducts. No enlarged glands. Inspection of the ducts and neighborhood revealed no abnormality. Single cystic artery lying to the liver side of the duct and parallel with it. Cholecystectomy from below upward. Appendectomy.

Macroscopical examination: Cholecystitis catarrhal subacute. Appendicitis chronic. The gall-bladder was normal in every particular except the mucous membrane, which was slightly edematous, uniformly pinkish in color, velvety to the touch. The appearance was properly comparable to a slightly granulated eyelid. The stones are worthy of mention. In number 40, varying in size from pin head to almost microscopic, in color brilliant chrome yellow, in form each, even the smallest, a perfect mulberry type, in consistency moderately friable. They were the smallest perfect stons I have ever seen.

Microscopical: (Dr. T. H. Dexter). Moderate hyperplasia of the muscular coat. Moderate infiltration of the mucosa with lymphocytes. Slight elevation of villi.

After course: Uneventful. Wound soundly healed 12.26.15. Patient has remained free from symptoms 6.1 .16.

Comment: This case I believe to be also one of gall-stones in a very young period. Their extremely small size yet perfect formation is of particular interest.

THE EARLY DIAGNOSIS AND TREATMENT OF CHRONIC NEPHRITIS

By JOSEPH H. BARACH
Pittsburgh, Pa.

The title of my discussion was purposely designated "Chronic Nephritis" because, as our knowledge stands to-day, during the life of the patient, we are unable to differentiate or diagnose the anatomical picture that is found post-mortem. Broad and narrow, hyaline or granular casts, a trace or cloud of albumin, high or low blood pressure, these individual evidences do not carry the far reaching diagnostic significance that we formerly attributed to them.

In a comprehensive review of the subject by Janeway, (1) we find that he considers the subject under four heads:

1. Patients in whom albumin and casts are the only evidence of the disease.

2. Patients with hypertension with or without a trace of albumin.

3. Patients with hypertension and outspoken myocardial insufficiency.

4. Patients with advanced renal insufficiency and uremic symptoms, superimposed upon one of the foregoing types.

It is readily seen if we allow our minds to run over these four groups that a single case may run the entire gamut of the classification. Rather is this classification applicable to the majority of cases as they come to the medical man for diagnosis and treatment, than the separate existence of four classes of nephritis cases.

The greatest proportion of cases do not come to us particularly early in the disease, although as time goes on, they are coming earlier. Insurance examinations, blood pressure estimations and constant discussion of nephritis as a medical topic have attracted the attention of the medical man; while through the interest of life insurance companies, their advertising and the offering of free examinations, have attracted the layman, with the result that a large number of persons who consider themselves well, have become acquainted with the importance and advisability of urinary and physical examinations. From all this much good will come.

Since as it is generally conceded there is no essential cure for nephritis and as we all very well know nephritis is the grim reaper of middle life, which carries away a large percentage of men who are doing the big share of the world's work, and carrying them off at a time when their experience and judgment is most valuable to society, a brief consideration of some of the principles involved—in the cause and recognition of the disease—may prove of some interest and benefit. Just how common nephritis is at the present day may be gleaned from the findings of the Life Extension Institute of New York. They find that in the State of New Jersey the death rate due to kidney and urinary diseases has increased 43 per cent., while heart diseases have increased 29 per cent., apoplexy and diseases of the nervous system 19 per cent. and so on.

Beyond a doubt the longevity of man should be increased with our better knowledge of hygiene, but it seems that our great desire "to live better" overbalances the good that should normally come from our knowledge of "how better to live."

But these are futile words to utter before physicians; and what concerns us mostly is how we may do our share to protect our patients against nephritis, and how much is it in our power to do so. The practitioner of medicine preaches much, and sometimes his patients take heed. I doubt if the medical profession will ever succeed in doing away with the occurrence of those cases of nephritis which come to the men and women who constantly overindulge in food, and I doubt if we shall ever succeed in teaching the great mass of people to dress properly and how to avoid body exposure, just as I doubt if we shall ever succeed in thoroughly acquainting our patients with the evils which accrue from alcoholism.

But there is a field, and a very large field, for good work, in the prevention of nephritis, and that is, for the physician to make it an iron clad rule of his practice never to discharge a patient who has had an acute infection until he has made sure that the patient's urine does not contain albumin and casts.

My own impression is, and I may be right or wrong, that in the majority of cases the general practitioner does not carry out the simple routine in all cases. I believe that it is our duty in all cases and we are honor bound to tell a man that he has not yet recovered and is not well, if his urine shows albumin and casts.

I would even go further and venture to say that a large proportion of the cases which later on we recognize as chronic nephritis and the etiology of which appears doubtful to our minds, have their origin in, or simultaneous with, acute colds, tonsilitis, pharyngitis, influenza, etc., to say nothing of the severer infections.

Attention may be called here to the recent findings of the streptococcus as the etiologic factor in the last epidemic of grippe: how pathogenic the streptococcus is for the kidneys is at once realized upon mention of scarlet fever, which so far as we know is a streptococcus disease. On the other hand, typhoid fever is an acute infectious disease in which I would say that nephritis is one of the rare complications. In a careful study of 500 cases of typhoid fever of which I have records of urinalysis during and after the disease, I found only one case in 427 leaving the hospital with albumin and casts in the urine.

When the patient with presumably chronic nephritis presents himself there are certain criteria by which we may establish or eliminate the diagnosis and some of these I will mention.

Evidences in the urine:

1. Incidental albuminuria of health: In the great majority of healthy persons undue physical exertion may be followed by albumin and casts in the urine for at least a brief period. Undue physical exertion, whether it occurs in the man who ordinarily lives a quiescent life or in a highly trained athlete; extraordinary exertion in any individual may bring on an albuminuria; and this albuminuria, I believe, is the result of a disturbance in the blood flow through the kidneys, and I have shown that it occurs most markedly where there is greatest disturbance in the pulse pressure. In practice we find that incidental albuminuria occurs frequently after such exertion as riding horseback, after running, dancing or skating. Incidental albuminuria also occurs after over-eating, over-drinking and exposure, and particularly when all three factors are combined. I mention this form of incidental albuminuria as the one which we may meet with in men and women who by their age, physique and predisposition would be candidates for chronic nephritis and who come to us presenting albumin and casts which disappear after a few days of proper regimen.

2. Febrile albuminuria, occurring after one of the acute infectious diseases is another form, which we are called upon to differentiate from the albuminuria of an established nephritis. Especially when the patient has not been aware of a pre-existing albuminuria.

3. Continued presence of albumin and casts: Only occasionally are we called upon to determine the kind of albumin present, for in the great majority of cases we are dealing with true serum albumin. More frequently do we meet with patients in whom the urine contains the true serum albumin, but their disease belongs to the orthostatic type. This type of albuminuria occurs in young persons. Here our individual is frequently of the asthenic type and gives no history of any apparent cause for nephritis. This orthostatic albuminuria is not at all rare: for in my own work I have come upon five typical cases during the past year. Our diagnosis in this type of cases is based upon the absence of albumin in the night urine, and an increased presence of albumin when the patient maintains the enforced erect posture for the period of an hour or longer.

Specific gravity:—A high specific gravity is evidence of the ability of the kidney to concentrate urine, whereas a persistent low specific gravity is evidence of the inability of the kidneys to concentrate urine. Normally the night urine is of darker color and higher specific gravity than the day urine. In advanced chronic nephritis and particularly in hypertension cases, we usually find the specific gravity of the night urine nearly the same as the day urine. The kidneys in such cases are excreting to their utmost throughout the twenty-four hours.

Elimination of salt and urea:—In recent times as the result of the study of functional tests, estimation of the amount of salt and urea eliminated daily has again become of diagnostic importance. In health, elimination of these substances varies with the intake, but in disease these substances are eliminated according to the character and extent of the damage done to the renal tissue. Not in all instances, however, is the non-appearance of the chlorids or urea in the urine an evidence of the inability of the kidneys to eliminate. There are times when these substances are retained within the body for some unknown reason, although the kidneys are quite capable of eliminating them. Certainly is this true in the chlorid retention of pneumonia, and as I have frequently seen in the course of nephritis, in which there is considerable variation from time to time, irrespective of the salt or nitrogen intake. Although the modus operandi of salt and urea excretion is not entirely understood, the ability of the kidneys to eliminate these salts serves as a valuable therapeutic guide.

Salt:—On a mixed diet the average normal individual will eliminate 10 grams or more of salt per liter, according to the intake. In chronic nephritis salt elimination is interfered with and this impermeability to salt often serves us as an early evidence of failure of function.

The salt test:—After determining the level of salt excretion by quantitative analysis on a fixed diet, the patient is given 10 grams of sodium chloride. When the excretory function is normal, practically all of the added salt will be excreted in the following twenty-four hours.

In a large number of cases salt retention and edema are directly associated.

Urea:—Urea exists in the blood normally in from 0.18 to 0.28

gram. per liter. In the developed nephritis when the kidneys are beginning to show inability to excrete the substance, the blood urea increases proportionately to the renal damage. In the urine in health, urea occurs in the proportion of about 15 grams per liter, which represent about 90 per cent. of the nitrogen intake. When an added amount of urea is ingested as in the urea test, nearly all of it is excreted within twenty-four hours unless renal function is impaired. McClain[3] has recently emphasized the value of Ambard's coefficient, the Urea Index. By collecting the urine over a given period and estimating the urea content of the blood at the mid-point of the period, we deduce the rate of urea excretion. McClain illustrates the diagnostic and prognostic value of this method.

Water:—While in health, the kidneys respond promptly in elimination of water, in nephritis the response varies. Water retention is frequently not recognized until late in the disease. The kidneys in a normal person will eliminate 500 c.c. of water within one to two hours after ingestion.

Martin Fischer[5] advises that the water test will be given on an empty stomach, preferably at about 8 A.M. Apropos of this I would like to mention that it has been my repeated observation that many persons who gain weight at the spas and lose it so quickly upon their return home have simply water-logged themselves with excessive drinking and their gain in weigh is rather an evidence of renal impermeability than of improved nutrition. Failure to eliminate water precedes the inability to eliminate urea.

Phenolsulphonphthalein:—The elimination of this substance has been studied very extensively and carefully and the consensus of opinion is, that elimination of sulphonphthalein by the kidneys is a valuable index of the functional ability of the kidney for the time of examination, and a positive proof only of the ability of the kidney to eliminate phenolsulphonphthalein. A single examination can only give a limited amount of information. All considered, nevertheless, the sulphonphthalein output is one of the best indices of renal function. The normal kidney after an intra-muscular injection will begin to excrete this substance in about 12 minutes and will eliminate from 40-60 per cent. the first hour; 20-25 per cent. during the second hour.

Renal test diet:—If we were to begin to study the renal function of a patient, spending three days in bringing the patient's excretions to an equilibrium, then one day for the salt test, one for urea, blood urea, sulphonphthalein, water and perhaps lactose, it is readily seen that considerable time is required, and only the very sick patient or the unoccupied man or woman will lend themselves to such observations. It was, therefore, a step forward when Hedinger and Schlayer proposed a renal test diet in which one day's rations contained a definite amount of water, nitrogenous foods and salt. This test diet has been adopted and slightly rearranged by Mosenthal.[4] Mosenthal's test diet can be easily arranged and is perfectly suitable for the great majority of cases. With a healthy kidney, the concentration of salt and nitrogen is usually above 1 per cent. and the total amount eliminated varies with the intake. Another advantage of this diet is in the fact that it simulates the normal demand upon the kidneys, and it seems to me that this is better than the administration of either salt, urea, or water alone on successive days.

Urinary acidity:—Stimulated by the claims of Martin Fischer and his predecessors, that a large proportion of nephritis cases are the result of acidosis, considerable interest has been aroused and much investigative work followed. The acidosis theory is also being applied in other fields of which Crile is the greatest exponent at the present time.

While some have brushed this interpretation aside, there is accumulating a large amount of incontrovertible evidence which warrants our serious consideration. That an acidosis exists in the uremia of nephritis is proved beyond a doubt. A striking clinical proof of this is evidenced by the Alkali retention test. In health the urine becomes alkaline after the ingestion of 5 to 10 grams of sodium bicarbonate, whereas in the acidosis of nephritis it may require 15 to 25 grams of sodium bicarbonate to render the urine alkaline. Thus the amount of sodium bicarbonate neutralized in the blood serves as an indication of the acidemia. Other evidences are to be found in the alveolar CO_2 and the bicarbonate content of the blood.

The urinary acidity should be measured in terms of hydrogen-ion acidity, and a valuable clinical observation, which I have seen borne out in advanced nephritis, is that when a patient's urine cannot be made alkaline to the point of reacting alkaline to thymolphthalein which represents an hydrogen-ion acidity of H," that patient is seriously ill and will not likely recover; Fischer claims that such patients never recover. Fischer also advises that the administration of alkali be not carried beyond the reaction point to thymolphthalein, saying that beyond that point the alkalies may do harm and aggravate the albuminuria.

Hypertension:—Here I will be very brief but very definite. A maximum blood pressure of 150 or over is abnormal. A minimum blood pressure of 100 or over is abnormal; both of these are serious indications. For ascertaining the degree of strain upon the cardiovascular system which the hypertension incites I make my estimations according to the Energy Index.[6] By adding the pressure which exists during the systole with that which exists during diastole, we have the total pressure per pulse beat, this multiplied by the number of pulse beats gives us the total pressure in mm. Hg. per minute. In normals we find that the S. D. R. Index seldom exceeds 20,000 mm. Hg. A high index indicates cardiovascular strain and overwork, a falling index indicates circulatory failure, or it may indicate that renal function has so improved that the strain upon the cardiovascular system has been removed.

Retinitis is occasionally a comparatively early symptom. The retinal changes may vary from a slightly increased prominence of the vessels, tortuosity, and a whitening of the vascular margins to the severe grades of extensive retinitis. I believe that in the future we shall have fewer cases in which the ophthalmologist will be the first to diagnose the disease. Prognosis upon retinal findings is often correct but it has been my experience frequently that the general condition of the patient as determined by functional studies and blood pressure readings warrants a much better prognosis than one to three years.

Treatment:—For the acute exacerbations of chronic cases, rest in bed, a limited diet low in salt and nitrogen, and purin-free. In the chronic cases, allow liquids in proportion to the ability of the patient to eliminate water. Arrange the patient's diet according to the need as predicated by the body weight, permitting a total caloric value commensurate with the needs of the individual. If the patient eliminates nitrogen well, we need not eliminate meat from the diet, if elimination of salt is not impaired, we need not insist upon a salt-free diet. Those food substances, the glands of the animal body, such as liver, kidney and sweet breads, those vegetables such as asparagus, mushrooms, peppers and the meat condiments and spices, all such foods which are rich in purins, extracts and irritants, should be forbidden.

Reduction in body weight is practically always accomplished by a fall in blood pressure. After the lower level of the first fall is reached, a further lowering of blood pressure is accomplished with considerable difficulty if at all. A very important point is, when shall we stop reducing the weight of the patient?

I have learned by repeated observation that we may reduce a patient's weight only to a certain point, with benefit to the patient; beyond that point, many patients begin to fail rapidly. It therefore seems advisable not to reduce the body weight after blood pressure has reached its lowest level. Elimination by the skin and bowels is a part of the routine treatment.

Treatment of urgent symptoms:—Edema may be relieved by restriction of the water intake or by increasing the water output through diuresis, diaphoresis and hydragogue cathartics. Usually we apply all these measures simultaneously or successively to the point of obtaining the best result. We administer digitalis or one of its derivatives, not so much for its diuretic effect as for its effect upon the cardiovascular system.

Restriction of chlorides:—The consensus of opinion to-day is in favor of restriction of chlorides to prevent edema. Opposed to this are the views held by Martin Fischer, who holds that hypertonic solutions of salt, glucose and soda cause dehydration of the swollen cells of the kidney substance and promote dehydration of all the body cells, thus relieving edema. While in the majority of cases we have, heretofore, seen edema disappear upon the removal of salt from the diet, I have carried out this Fischer treatment in a few cases of extreme edema of the legs, abdomen and scrotum with striking results. At present I am unable to state in which cases this method of treatment is indicated; but that it acts remarkably in some cases at least, I and some of my colleagues can affirm without hesitancy.

Urgent symptoms:—For the uremia with cerebral symptoms, hypertension dyspnea and irregular heart action, phlebotomy should be resorted to, taking 12-18 ounces. These patients quiet down, breathe easier, sleep, the pulse improves and cyanosis disappears. For the troublesome tracheitis of uremia I have found gram doses of bicarbonate of soda act better than morphine, codeine or inhalations, which are frequently used. This tracheitis seems to be the product of the acidosis which exists in the last stages of the disease.

For the insomnia in many cases we are forced to use morphine, although it leaves the patient depressed and affected the next day. In some cases, nitroglycerine at bed time will be conducive to sleep.

In the uremic stage in some cases we also have troublesome mental cases to deal with. There are perversions of smell or taste and at times we meet with actual syndromes of mental symptoms extremely suggestive of various types of mental disease, but fortunately these are of short duration.

REFERENCES

1. Janeway, Theo. C., Am. Jour. Med. Sci., Feb., 1916, Vol. CLI, No. 2.
2. Barach, Jos. H., "Physiological and Pathological Effects of Severe Exertion," Arch. Int. Med., April, 1910, Vol. 5, pp. 382-405.
3. McClain, "Urea Index," Jour. A. M. A., Feb. 5, 1916.
4. Mosenthal, Arch. Int. Med., Nov., 1915.
5. Fischer, Martin, "Edema and Nephritis," Wiley and Sons, Second Edition.
6. Barach, Jos. H., Jour. A. M. A., Feb. 14, 1914, Vol. LXII, p. 525.

VASCULAR VERSUS INTERSTITIAL NEPHRITIS

MAY THE DIFFERENTIATION BE MADE BY THE URANALYSIS?

BY B. G. R. WILLIAMS
Paris, Illinois

Frank interstitial nephritis with its characteristic symptoms and urinary findings and with its rapid and hopelessly progressive course, is a rare disease. Vascular nephritis with its many syndromes, variable and confusing urinary findings and a prognosis which damns the prognostician, is a very, very common disease.

This brief communication is intended as a warning to the man who attempts to predict the outcome of a chronic nephritis upon the basis of a single or an occasional uranalysis rather than a very close clinical and laboratory study of the case. Such a warning is needed because almost daily I see the diagnosis of interstitial nephritis where the case is one of arteriosclerotic kidney. This error tends not only to lead the physician into deep water in the special case under consideration, but to confuse medical literature and statistics to such an extent that the reader is perplexed when reviewing case reports.

It may be possible in a case of interstitial disease to intelligently diagnosticate and prognosticate by symptoms and a single uranalaysis. It is never possible to do so in vascular nephritis. During the past few years I have made this mistake myself, and have seen it made by some very clever diagnosticians. It is especially easy to err in cases where there is no hypertension, but where the arteriosclerotic process is limited to the blood vessels of the kidney. Be very reluctant to make a diagnosis of interstitial disease, seems to me to be good advice. In texts, you will find the statement that the two conditions cannot be differentiated by the uranalysis. However in a study of a series of urinary samples and with the following observations which I have tabulated, it may be possible in many cases to discriminate very accurately between interstitial and vascular nephritis:

TRUE INTERSTITIAL DISEASE	VASCULAR NEPHRITIS
AMOUNT OF URINE.—Persistent polyuria.	Varies in amount. Sometimes during the course of the disease there is more or less polyuria. Sometimes the amount is normal. Usually there is oliguria during a crisis. (See footnote.*)

---

*The term crisis may not be a good one, but I have used it to explain the peculiar symptomatology of vascular nephritis. A man in apparently good health suddenly develops symptoms more or less grave, and a uranalysis at this time shows albumin in large amount, granular casts, kidney cells and a paucity of solids. Three or four days later it may be difficult to detect albumin, and casts are found with some difficulty, although the solids may remain somewhat low. The urine, which may have dropped to less than a pint, rises to or above normal. A week later the patient refuses to remain in bed and, with the exception of slightly deficient solids or a questionable trace of albumin, no fault may be found with the urine. He may remain up and active for weeks, months or even years, with relapses and recoveries; but eventually he goes to bed for the last time. The "crisis" may be understood, it seems to me, by a brief pathological consideration. The peculiar intermittent attack in vascular nephritis is due to the fact that in the process the glomerular tuft is involved, and upon the destruction of this there is degeneration and loss of the subtending tubule.

SPECIFIC GRAVITY. — Low. Usually persistently very low.	Usually about normal or slightly low between the crises. Rarely normal during a crisis, but is usually low; more rarely high.
REACTION. — Usually slightly alkaline.	Rarely normal. Early in the course of the disease is usually excessively acid. Later there is persistent and marked fixed alkalinity. (The reaction, therefore, appears to furnish a crude prognostic index.)
ALBUMIN. — Usually slight amount. Amount remains stationary or almost so.	Slight or absent between crises. During a crisis, tremendous quantities may be present.

CASTS, DEGENERATIVE GRANULES, RENAL CELLS, ETC.—(The same observations as noted with albuminuria hold to a more or less extent. In interstitial disease, kidney débris remains stationary in amount or slowly increases. Epithelial shower is commonly noted as the fibrous tissue contracts, and usually mean a very early death. In vascular disease, microscopy is practically negative between crises. During the crises, kidney débris is present in large amounts. Cylinder shower means a "flushing out," and is of good prognostic meaning for that respective crisis, unless it chances to be the final crisis.)

When several of these units are simultaneously destroyed, the amount of urine is decreased and contains the products of the retrograde process—casts, albumin, etc. Coincidentally the functional equilibrium is disturbed and uremic symptoms appear at the same time the protoplasmic detritus is noted in the urine. A few days of rest enable the patient to compensate in part the functions being assumed by other tubules, which we know are able to meet this responsibility to a certain degree. At length the débris of the degenerative process is entirely washed away and the urine remains but slightly or not at all abnormal. Later he may lose another portion of his renal parenchyma, may again compensate, and so on until he has no more renal cells to spare. Although I have never seen the above explanation proposed, I have become convinced that such is the process, and have applied the above principles with considerable satisfaction to those cases I have studied during the past few years.

CONCLUSION

The above table and the observations recorded in the footnote have been based upon a personal study of about 20,000 urines (19,682 specimens at the time of writing) which were not norms, but where renal disease was suspected.

Avoid the diagnosis of interstitial disease where it is possible to diagnosticate vascular nephritis. Base the latter decision not upon the blood pressure readings, but upon a series of uranalyses checked by the symptomatology. Attempt if possible to recognize the stage which I have termed the "crisis" and to comprehend its meaning. Avoid a prognostic opinion in any case of vascular nephritis (except perhaps in aged persons with an extended history—but even here I have seen a man of 75 recover and return to his desk for two more years). Even though he be panting and nauseated, even though he lie in coma and even though the urine be passed by the cubic centimeter and be saturated with albumin or loaded with casts, or be passed not at all, do not commit yourself to an opinion.

Finally it does seem to me that the time has come when most cases of vascular nephritis should be recognized by the diagnostician and no longer confused with interstitial disease. During the past years the laboratory man has not done his part, but the main responsibility has fallen upon the blood pressure apparatus and the prosector.

THE RELATION OF MIGRAINE TO THE SO-CALLED ACIDOSIS OF CHILDREN

By JOHN A. LICHTY
Pittsburgh, Pa.

A common error is to interpret attacks of periodic vomiting in children and infants as being attacks of so-called acidosis when they are in reality precursers of a definitely established course of migraine. Acidosis or cyclical vomiting in children has received a great deal of consideration in the past decade. Various interpretations have been given to explain the cause of acidosis. Zade (Arch. f. Kinderheilkunde, July 25, 1914) thinks "It is due to sudden psychogenic disturbance of carbohydrate metabolism together with irritation of the vomiting center" wherever that may be located.

Such an explanation might well do to account for the cause of migraine. It is not any more definite than many other causes which have been suggested. Kerley (Am. Jour. Dis. Child., Oct., 1914) found that "rheumatism" occurred in one or both of the parents in about one-half of the cases of children suffering from acidosis, and sick headaches occurred in the parents of the remainder of the acidotic children. In my limited experience with cyclical vomiting in children I have nearly always found a hereditary history of migraine in one or more of the forebears. In some of these children I have had the rare privilege of seeing the transformation from cyclical vomiting to typical sick headaches.

The term acidosis is not comprehensive enough to cover the condition of so-called cyclical vomiting. It is only a symptom which may arise in any case where a shorter or longer period of starvation has existed. This is well brought out in a paper by Howland and Marriott entitled "A Discussion of Acidosis" with special reference to that occurring in diseases of children (Bull. Johns Hopkins Hospital, March, 1916). They say "We may in many instances liken the mere presence of acetonuria to fever, for it occurs in most of the infectious diseases of children with much the regularity that fever does. We do not look upon moderate fever associated with infectious processes as unusual or dangerous. Hyperpyrexia may however develop and in itself be dangerous or fatal. So too a production of the acetone bodies may in itself determine a fatal outcome." In the protracted nausea and vomiting which are occasionally associated with an acute peptic ulcer especially when located in the duodenum acetonuria is not infrequent, and will disappear as soon as feeding by the mouth can be resumed. Also in cases where starvation must sometimes be instituted or rectal feeding established, acetonuria nearly always occurs. One of the strong arguments against the inadequacy or uselessness of rectal feeding is the invariable appearance of the acetonuria of starvation. For these reasons one may well conclude in the case of cyclical vomiting of children with the usual acetonuria, that the acetone is not the cause of the condition, but the result, and I wish to go further by stating if one will take the time to inquire into the history of these little paroxysmal sufferers he will find in the large majority of cases a satisfactory history of migraine.

I will here report a few cases illustrating the point:

Case I. The patient who is now a lad of thirteen has been under my observation since his birth. The mother before she gave birth to this, her only child, was remarkably neurotic, at times hysterical, and again manifesting grave neurasthenic symptoms. She had the auræ of migraine but I had not recognized them as such. Her mother, that is, the patient's grandmother, had attacks of migraine. After confinement the mother lost her neurasthenic and hysterical symptoms and had definite paroxysmal attacks of migraine. The patient as a little baby had paroxysmal attacks of nausea nd vomiting almost from birth, certainly during the first year of life. The attacks corresponded closely with the description of attacks of cyclical vomiting or so-called acidosis. At times they were quite grave and there was fear of a fatal termination. At the age of ten the patient began to have one-sided headaches associated with vomiting and later the headaches appeared as typical attacks of migraine.

Case II. Miss H. M. S. act. 5 yrs. has attacks of vomiting which come after irregular intervals. Between attacks is well. Acetone found in the urine during attacks. With one of her attacks had headaches. Father and grandmother on father's side and also an aunt on mother's side have migraine.

Case III. Miss O. W. D. act. 4 yrs. complains of attacks of pain in abdomen which come on every two weeks. Has had these since the age of ten months. Recently has vomiting with the attacks. Between attacks is well. The mother has severe left-sided sick headaches and I expect this little girl later to have sick headaches.

In addition to these three patients I have seen possibly half a dozen more who had so-called cyclical vomiting, later called acidosis, and now have fully developed attacks of migraine. In seven hundred cases of migraine I have obtained the history every now and then of bilious attacks with obstinate vomiting in childhood with migraine coming on at puberty. No doubt acetone occurred in the urine of these patients when they were for some time without food, and a condition of acidosis might have been said to exist. However, it is best to conform with the principles which Howland sets forth in his paper before a diagnosis of acidosis is made. I think it well to search diligently for a hereditary history of migraine before interpreting a condition of acidosis in childhood or before the age of puberty.

CLINICAL DIAGNOSTICS OF DISEASES OF THE HEART

BY EDWARD E. CORNWALL
Attending Physician, Williamsburgh and Norwegian Hospitals; Consulting Physician, Bethany Deaconesses Hospital
Brooklyn-New York

The brief presentation of some of the more important points in the clinical diagnosis of diseases of the heart which is here given, is intended for the general practitioner, and deals only with topics which lie within his ordinary scope; and it avoids discussion of topics and methods of examination which belong more particularly to the specialist and research worker and the laboratory.

The subject, as it presents itself for discussion, shows two sides, that of the individual symptoms and signs, and that of the particular diseases; and divides naturally into general diagnostics and special or differential diagnosis.

GENERAL DIAGNOSTICS

General diagnostics deals with the significance of the individual facts, symptoms and signs which are elicited at the bedside by interrogation of the patient or others regarding his family and personal history; by inspection, palpation, percussion and auscultation of his body; by the use of special instruments to increase the examiner's sense perception, and by the use of special tests.

HISTORY

The facts of *family history,* which should be investigated in diseases of the heart include not only those which refer to heart disease itself, but also, in particular, those which refer to rheumatism, gout, obesity, nephritis and apoplexy. A tendency to these conditions is hereditary to a certain extent, and their relation to heart disease is tolerably distinct.

The patient's *personal history* may be divided, for convenience, into two periods, that before the beginning of the present illness, and that after.

The *previous personal history* should be searched for diseases and conditions which are known to be possible or probable causes of heart disease, notably rheumatic fever, scarlet fever, pneumonia, influenza, typhoid fever, syphilis, gout, lead poisoning, septic conditions, excessive use of alcohol, tobacco, tea or coffee, excess in eating, physical and mental overstrain, emotional disturbances, and derangements of the gastrointestinal tract; also for symptoms which the patient may have noticed in the past, which are suggestive of heart disease; also for occupations and incidents which can predispose to or cause heart disease; and also for diseases in which affections of the heart may occur as secondary manifestations.

The *history of the present illness* consists in a chronological account of the patient's symptoms.

SYMPTOMS

Dyspnea is the most important subjective symptom of cardiac insufficiency, particularly when observed only after slight physical exertion. Then it is characteristic of an imperfectly compensated valvular defect or of weakness of the myocardium from any cause. Paroxysmal dyspnea may occur in heart disease, though it also occurs in other conditions which must be differentiated, such as dyspepsia, bronchial asthma, chronic nephritis, and pressure of a tumor or aneurism on the pneumogastric nerve. Orthopnea usually indicates an advanced stage of decompensation in valvular disease, though it may occur in purely functional disorders of the heart.

In congenital cardiac disease dyspnea may be extreme, but more often it is slight or absent altogether, even when cyanosis is marked.

In acquired mitral stenosis or incompetence dyspnea is a common symptom, and one of the earliest to indicate failing compensation. It is due to passive congestion of the lungs. In cases with badly ruptured compensation it may be so great that the patient is unable to breathe easily lying flat; he breathes more easily in the sitting than in the prone position, because when his body is vertical the passive congestion in the medulla is less.

In lesions of the aortic valve dyspnea is not so prominent a symptom as in lesions of the mitral valve, and when it does occur it is not due to passive congestion of the lungs or of the medulla, unless there is also a mitral leak, but to ischemia of the medulla, and in rare cases to irritation of the vagus by periaortic inflammation, which may induce asthmatic attacks.

In lesions of the pulmonary valve dyspnea is not usually a pronounced symptom even after severe exertion. When it does occur it may be due to an accompanying tuberculosis.

In pericarditis, plastic, effusive or adhesive, dyspnea may be severe, slight or absent, depending on the quantity of exudate, the rapidity with which it has formed, and the condition of the heart muscle, as well as on pulmonary or pleuritic complications or irritation of the vagus. Large exudates may cause pressure on the superior vena cava, the lungs or the bronchi, or cause downward displacement and fixation of the diaphragm. Pericardial adhesions cause dyspnea by interfering with the action of the heart, weakening its muscle, and producing cardiac insufficiency.

Secondary pleural effusion may aid in producing dyspnea.

In acute degeneration of the myocardium caused by the infectious diseases dyspnea may occur if the cardiac insufficiency becomes pronounced. During convalescence from the infectious diseases dyspnea occurring from slight causes is significant of myocardial degeneration.

In fatty heart and all forms of myocardial degeneration dyspnea on slight or moderate exertion is a regular and prominent symptom, and the one which is usually complained of first. In thrombosis of the heart the dyspnea is usually intense, although exceptionally it is slight.

The form of dyspnea called cardiac asthma, which occurs in well marked paroxysms, is observed most frequently in aortic insufficiency and coronary solerosis. The patient is awakened by the need for air caused by accumulation of carbon dioxid in his blood; and the latter probably also weakens his heart; so the attack may be due to acute failure of the left ventricle as well as to failure of respiration. In any case other conditions producing asthma may be present and be the exciting cause.

The variety of dyspnea known by the name of Cheyne-Stokes breathing may occur in an advanced stage of cardiac insufficiency; it is usually of grave prognostic significance, but not infrequently it disappears after lasting for a considerable time, even weeks.

Cough is a common symptom of cardiac insufficiency, and is due to passive congestion of the lungs. It is often an early sign of ruptured compensation in valvular disease. In some cases of pericarditis it is a prominent and distressing symptom.

Hemorrhage from the lungs, nose, stomach, or uterus sometimes occurs in heart disease as a result of passive congestion. From the lungs such hemorrhage occurs most frequently in mitral stenosis.

Coldness of the extremities is found in nearly all forms of heart disease, and is due to poor circulation.

Pain in the region of the heart is characteristic of angina pectoris, and may be intense and agonizing, or it may be moderate or anginoid in character; and it may be reflected to the shoulders and down the inner aspects of the arms. Pain is often present in the early stages of pericarditis. It is common in inflammatory conditions of the aorta. It is frequently met with in the cardiac neuroses and in dyspepsia, and in chronic poisoning with tobacco and coffee. It is rare in disease of the myocardium or in chronic valvular disease, with the exception of mitral stenosis. It may be present in acute dilatation of the heart.

Palpitation of the heart is usually due to nervous causes; it is a common symptom in neurasthenia and hysteria; it is often found at the time of puberty; it occurs very frequently in dyspepsia; it sometimes appears as a symptom of intestinal toxemia; the abuse of alcohol, tobacco, tea or coffee often produce it; it may occur as a reflex from the pelvic organs; and it may also occur with organic disease of the myocardium or valves.

Sensations of irregularity or intermission of the heart's action, (skipped beat,) are usually due to nervous conditions; and the cardiac irregularities which produce the sensation (often extra systoles or hemi systoles), may persist without necessarily indicating serious organic disease. But this symptom, in the presence of other symptoms and signs of organic heart disease, may support or strengthen the diagnosis.

Sensations of pressure or tightness in the precordium may indicate organic disease of the heart or aorta, but more often they seem to be due to nervous or emotional causes.

Pulsations felt in the head or neck are common in nervous and emotional conditions, but they may be due to overaction of the heart connected with organic disease.

Head noises are common symptoms in heart disease, and may be significant of organic disease of the heart or arteries, or both. They seem often to depend to a certain extent on the relative state of the blood pressure.

Yawning may be a continuous and distressing symptom in acute dilatation of the heart.

Nervous symptoms, such as headache, dizziness, faintness, delirium, hallucinations, loss of memory, insomnia, pain in the extremities, twitching of the muscles, languor and debility, may all occur in heart disease, but as they are common in other conditions their diagnostic value is slight.

While the family and personal history of the patient and his symptoms supply data of great value in making the diagnosis in heart disease, their value is, in general, inferior to that of the physical signs, which are elicited by inspection, palpation, percussion, auscultation, and the use of special instruments.

INSPECTION

Attitude and General Appearance

The attitude may suggest dyspnea in varying degrees. Panting on slight exertion and orthopnea are suggestive of heart disease. Emaciation is often found with valvular lesions which are not fully compensated, more particularly with mitral lesions and in children. Emaciation of moderate degree combined with a dry cough and dyspnea on slight exertion in a young adult, are particularly suggestive of an imperfectly compensated valvular lesion as well as of early phthisis, for which it is often mistaken. It happens too often that patients with mitral disease walk around with this condition imperfectly compensated, supposed to have consumption, until the rupture of compensation is complete. Clubbing of the finger ends is found in valvular lesions both acquired and congenital, but it is not pathognomic of those conditions, being also a common sign in chronic disease of the lungs.

Appearance of the Skin and Mucus Membranes

Cyanosis is distinctly characteristic of heart disease after we have eliminated conditions of the respiratory tract which mechanically interfere with the access of sufficient air to the lungs and blood. In infants and young children it suggests a patent foramen ovale or an aperture in the interventricular septum, or a congenital valve lesion; and in these patients it may be excessive in proportion to the dyspnea. In acquired valvular disease and chronic myocardial degeneration it appears when the circulation is markedly insufficient. It is often seen in the failing heart of pneumonia. It may be caused by the pressure of mediastinal tumors on the superior or inferior vena cava at their entrance into the heart.

Pallor is commonly found in young adults suffering from aortic incompetence. In infants it suggests the possibility of a pulmonary stenosis. After anemia and nephritis have been excluded the most likely cause of pallor is heart disease.

Jaundice which is slight, occurring early in heart disease, is probably due to catarrh of the bile ducts, but occurring late it points to organic changes in the liver produced by long continued passive congestion.

Edema beginning in the feet and ankles is of common occurrence in chronic cardiac insufficiency. When it extends beyond the lower extremities it usually invades first the scrotum and back, then the head and arms, and finally the subcutaneous tissues generally. In the slow failure of chronic myocardial degeneration the edema may be extensive and solid. Although edema may occur in all forms of cardiac insufficiency it is most commonly observed in mitral incompetence.

Bulging and Retraction of the Chest Wall

Bulging of the chest wall in or near the precordium is not uncommonly seen in heart disease. It is often observed as a result of enlargement of the heart following valvular disease in children, whose thin and elastic chest wall gives way more easily than the comparatively rigid chest wall of the adult. In adults, especially those past middle life, the enlargement of the heart must be very great indeed to produce a visible bulging, and when it does occur it is usually limited to the lower half of the precordial area.

A general pushing forward of the precordium with filling up of the intercostal spaces suggests pericardial effusion, especially if confined to the region on the left of the sternum.

Local bulgings above the level of the fourth rib are usually due to an aneurism.

Retraction or depression of the precordium, particularly in its lower half, is sometimes seen as a result of pericardial adhesions. In every case showing bulging or retraction, before ascribing a cardiac cause, we should exclude malformations, alterations in form due to disease or injury of the bone or cartilege, tumors, and disease of the lungs or pleura.

Abnormalities of the Apex Beat

The apex beat may be abnormal in position, extent, strength, or rhythm. These abnormalities in many cases may be seen, but they are often best felt; and in examining the apex beat palpation is usually combined with inspection. In many cases the apex heat is not visible at all, and in some can not even be felt; then our only method of estimating it is by auscultation. When faintly visible it can be best appreciated by an oblique inspection, or an inspection with the light thrown on the chest at an acute angle. It may be invisible in conditions of perfect health, especially in obese or thick chested or large lunged people; and it may be visible in the erect and invisible in the prone position.

The apex beat may be *changed from its normal position* in the fifth left interspace about three inches from the mid-sternal line, by the following conditions: Lying on the left side shifts it to the left one or two inches. Lying on the right side produces less displacement. A full inspiration lowers it about one inch. Hypertrophy and dilatation of the right ventricle displace it to the left. Enlargement of the left ventricle displaces it to the left and downwards. Pleuritic effusions may displace it to the right or left. Pericardial effusion, if considerable, displaces it upwards. Pleuritic adhesions or fibroid contraction of the lungs may displace it to the right or left. Tumors may displace it according to their position. Aneurismal enlargements may displace it downward. Distension of the stomach or peritoneal cavity may push it upwards.

Increase in extent of the apex beat, if moderate, is usually accompanied by displacement and increase in force. If the increase is considerable, so that the impulse involves two or three interspaces outside of the mammillary line, with a central point of maximum impulse, it indicates extensive dilatation and hypertrophy, with predominance of the latter; but if the impulse is weak and diffuse, dilatation predominates over hypertrophy. In cases of extreme dilatation and hypertrophy in children the lower half of the sternum with attendant ribs may rise with each ventricular systole. A moderate increase in extent and force of the apex beat without dislocation may be seen in functional disorders, after violent exertion or excitement, after excessive use of tea, coffee, alcohol or tobacco, and some drugs.

Diminution in extent of the apex beat or complete disappearance of it, may be seen in healthy persons with large lungs or thick chest walls; in emphysema with lungs overlapping the heart, in encroaching pleural and pericardial effusions, in pericardial adhesions, in myocardial degeneration, in shock, collapse, or any condition which greatly weakens the ventricular systole. It is sometimes seen in aortic stenosis when the systole is so slow and labored that the apex fails to make a decided impact against the chest wall.

Variations in strength of the apex beat are best appreciated by palpation, but they may to a certain extent be seen. The same is true of *variations in rhythm,* which often require for their proper appreciation combined palpation, auscultation and examination of the pulse.

Abnormal Pulsations in or Near the Precordium

Pulsations other than the apex beat are sometimes observed in or near the precordium, which possess more or less diagnostic significance.

In the second or third left interspace, near the sternum, pulsations may be due to retraction of the lung, to dilatation of the right ventricle and conus arteriosus and pulmonary artery uncovered of lung (often seen in mitral stenosis with compensatory hypertrophy of the right ventricle,) to dilatation of the left auricle, or to aneurism of the ascending portion of the arch of the aorta.

In the third, fourth and fifth left interspaces, between the sternum and parasternal line, pulsations may be due to hypertrophy and dilatation of the right ventricle, and, very rarely, to aneurism of the ventricular wall.

In the first and second right interspaces, near the sternal border, pulsations may be due to aneurism of the ascending portion of the arch of the aorta, especially if expansive and attended with swelling. It may also be caused by violent contractions of the left ventricle communicated to the aorta, or to exposure of the aorta when the heart is pushed or drawn to the right side.

In the second, third and fourth right interspaces pulsations may be due to the causes mentioned immediately above, or if presystolic in time, to a dilated right auricle.

In the third, fourth and fifth right interspaces, near the parasternal or mid-clavicular line, pulsations may be due to displacements of the heart to the right.

Over the manubrium sterni, pulsations point to eroding aneurism of the transverse portion of the arch of the aorta or of the innominate artery.

In the veins of the neck, if systolic in time, pulsations suggest tricuspid incompetence. Such pulsations are often seen in aneurism.

In the arteries of the neck, pulsation is a prominent sign in aortic incompetence and also in aneurism; and it is commonly observed in cardiac neuroses and in overaction of the heart from any cause.

Just above or below the clavicle, pulsations, if near the sternal end, suggest aneurism; if near the distal end, they are without diagnostic significance.

Near the left border of the heart, pulsations may be due to a tense collection of pus in a sacculated empyema, which transmits the cardiac impulse to the adjacent chest wall; or, by a similar action, to a vascular malignant tumor of the lung; or to a portion of solidified lung on close contact with the heart.

Epigastric pulsation, occurring temporarily, may be due to simple overaction of the heart caused by emotion or exertion; but when persistent it is usually due to some organic cause. It may be an evidence of displacement of the heart. Its most common cause is compensatory hypertrophy of the right ventricle. It is often seen in aneurism of the abdominal aorta, in which case it is post-systolic in time. It may be caused by a tumor lying over the aorta in the epigastric region which transmits the impulse of the aorta.

Capillary pulsation, made evident by pressing a thin piece of glass against the lip or finger tip, is sometimes observed in aortic incompetence.

Pulsation of the peripheral veins has been observed in aortic incompetence.

Diastolic collapse of the jugular veins is sometimes seen in pericardial adhesions.

Systolic retraction of the precordium generally suggests pericardial adhesions, but if limited to the fourth and fifth left interspaces in the region of the apex, it may also suggest hypertrophy of the left ventricle.

Systolic retraction of the epigastrium is a sign of mediastino-pericardial adhesions.

PALPATION

Palpation confirms or modifies the data obtained by inspection and reveals signs which cannot be appreciated by the eye. It gives us a fuller knowledge of the apex beat than can be learned by the eye alone. It often makes plain the nature of general or local precordial bulgings or depressions. It enables us to determine some abnormal pulsations that are not plainly visible. By it we appreciate thrills, friction fremitus, valve shocks, and sometimes fluctuations due to the presence of fluid in the precordium.

The apex beat

The diagnostic significance of abnormalities in the position and extent of the apex beat have been already alluded to in the discussion of the data given by inspection. Here are discussed the variations in force and rhythm of the apex beat, which are appreciated better by palpation than by inspection.

Variations in force of the apex beat, as felt by the hand, are found in health. The apical impulse is temporarily strengthened by inclination of the body forward and to the left and weakened by inclination to the right. It is increased after a full meal and by mental and physical exertion. It is relatively stronger in children than in adults because of the thinner chest walls. In old age it may be stronger because of senile hypertrophy of the left ventricle, or weaker because of senile enlargement of the lungs. It is weaker in persons with thick than in those with thin chest walls.

Persistent increase in force of the apex beat is most commonly due to hypertrophy of the left ventricle, in which case the apex is displaced beyond the nipple line, in the fifth or sixth interspace, and its area is moderately enlarged. It may also be increased in acute endocarditis or pericarditis, in pericardial adhesions where the lung is retracted, and in cases with consolidated lung overlapping the apex. It is sometimes increased by continued abuse of tea, coffee, alcohol, tobacco and certain drugs. It is increased in some of the cardiac neuroses.

A weakened apex beat or one imperceptible to the touch may be consistent with health, especially in the obese, or it may be a sign of myocardial degeneration, or of emphysema, or of fluid in the pericardium or pleura. Obstruction at the aortic orifice, by causing a slow, labored systole of the left ventricle, may be responsible for a weak apex beat.

Variations in rhythm of the apex beat can be studied most conveniently in connection with the heart sounds and the pulse, and they will be discussed in those connections.

Thrills

A thrill is a short, trembling sensation, something like that felt by putting the hand on a purring cat, but with finer vibrations. It is usually due to an organic valve lesion, although it may be due to the heart changes produced by anemia. When clearly made out it is of great diagnostic value. In examining thrills we should note the region in which they are felt and their place in the cardiac cycle. They may be systolic, diastolic and presystolic, the last being the most distinct.

In the *mitral area,* at or just inside the apex, and usually in a small field, a presystolic thrill is often felt, which ends abruptly with the impact of the apex against the thorax. It is pathognomonic of mitral stenosis. This thrill has a distinct purring feel with fine vibrations, or a hesitating, jogging feel; or it appears as a very short, rough vibration immediately before the apex beat. It is usually limited in time to the auricular contraction, but occasionally it occupies the greater part of the diastole. A systolic thrill in the mitral region is usually due to regurgitation through the mitral orifice, though rarely it is caused by aortic stenosis and transmitted to this region from the aortic orifice. A diastolic thrill due to aortic incompetence is very rarely felt over the apex.

In the *aortic* area a systolic thrill may be felt, which indicates obstruction at the aortic orifice; it is next in frequency of occurrence after the mitral presystolic thrill. It may or may not be transmitted down the sternum and toward the apex. A diastolic thrill observed in the aortic area, which may or may not be transmitted down the sternum and toward the apex, is produced by aortic incompetence.

In the *pulmonary valve area* a systolic thrill is regularly produced by pulmonary stenosis, and it may also be due to fibroid phthisis with retracted lung. A diastolic thrill in this region would indicate the rare condition of pulmonary regurgitation. A thrill felt in this region which persists throughout the systole and part of the diastole indicates a patent ductus arteriosus.

In the *tricuspid area* a systolic thrill may be due to tricuspid incompetence, in which case the diagnosis can usually be confirmed by the other signs of this condition. Such a thrill is more commonly a transmitted one from the aortic orifice. A diastolic thrill felt in this region is usually transmitted from the aortic orifice. A presystolic thrill felt in this region is extremely rare, and would indicate tricuspid stenosis.

Thrills felt in the *area between the level of the fourth rib and the episternal notch, within two inches of the sternal border on either side,* may be due to aneurism of the aorta, but the differential diagnosis between aneurism and valvular disease must be made by the other signs.

Friction fremitus

Friction fremitus is a superficial, rubbing sensation, coarser than a thrill, which has been compared to the feel of a piece of sand paper passed lightly over the palm of the hand. It has a to-and-fro character, and is not so closely connected with the normal heart sounds as are the thrills due to valvular lesions. It is found over the body of the heart, usually between the second and fourth interspaces, and indicates pericarditis or pleurisy with fibrinous exudadation; but it is not a very common sign in pericarditis: when present, its intensity may be increased by moderate pressure and diminished by forcible pressure.

Valve shocks

In persons with thin chest walls the closure of the heart valves is sometimes felt with exaggerated intensity. The diastolic shock of the pulmonary valve is the one most frequently observed, as a pathological sign, and indicates increased tension in the pulmonary artery due to mitral disease, or obstruction in the lung with overaction or hypertrophy of the right ventricle. Increased tension in the aorta may similarly produce a palpable shock over the aortic valve. Systolic shocks may sometimes be felt over the lower part of the sternum, usually in the fourth interspace near the left edge of the sternum, which are caused by forcible closure of the auricular-ventricular valves in cases with hypertrophied ventricles.

Fluctuation

A fluctuation wave may sometimes be felt in the *lower precordial* interspaces in cases with large pericardial effusion.

Tenderness

Tenderness may sometimes be elicited in superficial areas, which may be reflexes from cardiac conditions. Head has shown that disease of the aortic region and ventricles gives rise to pain and tenderness over the upper areas of the chest and the mid-orbital region of the scalp; and that disease of the mitral region occasions pain and tenderness in the lower part of the chest, the upper part of the abdomen and the temporal and frontal regions of the scalp.

PERCUSSION

By means of percussion we secure the most reliable information concerning the position, size and shape of the heart which it is possible to get by ordinary physical examination.

Area of cardiac dulness

It is customary to speak of areas of superficial and deep cardiac dulness, or of uncovered and covered dulness. The area of *uncovered dulness* corresponds to that portion of the anterior surface of the heart which is not usually overlapped by lung. It is approximately a triangle bounded internally by the mid-sternal line between the upper border of the fourth costal cartilege and the junction of the ziphoid cartilege with the sternum, and the two lines drawn from the extremities of this line to the apex. This area is diminished by deep inspiration, emphysema, pneumopericardium, air in the pleural cavities, stomach or intestines which displace the heart, pericardial adhesions which draw the heart under the lungs, and possibly atrophy of heart. It is increased by deep expiration, by pericardial effusion and by cardiac dilatation and hypertrophy. In cardiac diagnosis the determination of the area of uncovered dulness is of little practical value. In healthy persons it may be smaller or larger than usual owing to natural encroachment or recession of the lungs. For the purpose of determining the position, size and shape of the heart, examination of the area of covered or deep dulness is much more effective, and is the only one that need be considered.

The *outline of the area of covered dulness* corresponds very closely with the outline of the heart, whose relations to the chest wall are as follows: The upper boundary is a horizontal line drawn at the level of the third costal cartilege from a point one-half inch to the right of the sternal edge, to a point one inch to the left of the sternal edge. The lower boundary is a line drawn from a point in the middle of the junction of the upper and middle thirds of the ensiform cartilege to a point in the fifth left interspace, a half inch inside the mid-clavicular (usually nipple) line, or three and a half inches to the left of the mid-sternal line. It is bounded on the right by a line curving gently outwards, connecting the right ends of the upper and lower boundary lines. It is bounded on the left by a slightly convex line connecting the left ends of the upper and lower boundary lines.

Methods of percussion

The methods of percussion employed for mapping out the area of covered dulness are the simple, pleximetric, auscultatory and palpatory methods.

In *simple percussion* the middle finger of the right hand is used as a hammer, or an actual hammer with a rubber head is used; and the stroke is applied directly to the chest wall or to the middle finger of left hand laid on the chest wall, usually to the latter. Sometimes the middle and two adjoining fingers of the left hand are laid on the chest wall and the three fingers tapped seriatim. A variety of simple percussion called *threshold percussion* is distinguished by the lightness of the percussion stroke; when the border of the area of dulness is reached the light percussion stroke becomes inaudible.

In *pleximetric percussion* a pleximeter of wood, hard rubber or other hard substance is held against the chest wall by the index and middle fingers of the left hand, while it is struck by the middle finger of the right hand or a hammer.

In *auscultatory percussion* the examiner usually applies the stroke directly to the chest wall while he listens with a stethoscope to the percussion note. The bell of the stethoscope is held against the chest wall in the center of cardiac dulness while the percussion strokes are made in line toward it, or along vertical and horizontal lines; and the points where first a difference in the percussion note is perceived mark the outline of covered cardiac dulness.

In *palpatory percussion* the chest is patted or scraped with the palmar surface of the index or other finger.

A variety of palpatory percussion which has been described by the writer and called by him *feel percussion,* is performed as follows: With the tip of the finger of the right hand, the finger being bent as if for playing the piano and held loosely at the metacarpophalangeal articulation, and with motion mostly at the wrist, a series of very light touches or taps are made rapidly along a line on the surface of the body. Between these touches or taps the finger is raised from the skin just enough to separate the touches or taps from each other; and no sound is made by the impact of the finger tip on the skin. The operation looks like feeling for something; and that is what it is. What is felt for is a difference in density, which informs the examiner of the presence of solider masses: e. g., the cardiac edge.

It is not usually possible to delimit by percussion more than the right and left borders of the area of covered cardiac dulness, but that is enough for most diagnostic purposes.

Diagnostic significance of variations in the cardiac outline

Variations from the normal in the outline of the area of covered cardiac dulness have the following significance:

Extension downwards and to the left, with the apex beat displaced in the same direction, usually means hypertrophy and dilatation of the left ventricle.

Extension to the left beyond the nipple or mid-clavicular line usually indicates dilatation of the left ventricle.

Extension above the level of the third rib, if pericardial effusion be excluded, suggests enlargement of the infundibulum of the right ventricle, enlargement of the left auricle, aneurism of the aorta, or mediastinal tumor.

Extension chiefly to the right with a rounded apex usually means hypertrophy and dilatation of the right ventricle and auricle.

Extension both to the right and left, with a diffuse apex beat, indicates hypertrophy and dilatation of both ventricles.

Extension to the right, left and upwards, is a sign of pericarditis. The area of dulness in this case is more or less triangular in shape, with the base directed downward. A characteristic sign in pericardial effusion is increase in obliquity of the cardio-hepatic angle.

Extension to the right only, with enlargement of the cardio-hepatic angle, suggests the presence of inflammatory exudate or a neoplasm in the mediastinum.

AUSCULTATION.

The evidences of cardiac disease which we obtain by auscultation are the most valuable of all, and are often sufficient by themselves to enable us to make the diagnosis.

Auscultation is performed directly with the ear against the chest wall, or through the medium of the stethoscope. The latter method is the most convenient generally, but the former has its advantages, especially in cases with greatly dilatated ventricles, for the ear applied to the chest can palpate the apex beat at the same time that the sounds connected with the action of the heart are heard, thereby enabling the sounds to be more easily placed in the cardiac cycle. Ordinarily their place in the cycle is determined in doubtful cases by aid of the finger placed on the carotid artery, or, with proper allowance, on the radial pulse.

We listen in the precordial region for the regular heart sounds, which may be variously modified in disease, and for adventitious sounds which are connected with the action of the heart.

The valve areas

As most of the sounds connected with the action of the heart arise at the valvular orifices, we listen first in the valve areas. These areas are regions on the chest wall where the normal closing sound of the valves themselves are heard most distinctly. They are not situated immediately over the valves, and this fact renders the task of referring particular sounds to their proper valvular origin much easier than it otherwise would be; for the valves lie so close together in their projection on the surface of the chest that the bell of a large stethoscope can include some part of all of them. The sounds arising at the valvular orifices are transmitted to the surface along the lines of best conduction, which in every case lead away from the nearest surface point.

The *mitral area* is in the region of the apex. Here, in a space about one inch in diameter, the closure sound of the mitral valve is best heard.

The *aortic area,* where the closure sound of the aortic valve is heard most distinctly, is in the second right interspace near the sternal border.

The *tricuspid area,* where the element of the first heart sound due to the closure of the tricuspid valve is heard most distinctly, lies in the lower part of the sternum, near its left border, opposite the fourth and fifth interspaces.

The *pulmonary area,* where the closure sound of the pulmonary valve is most clearly audible, is in the second left interspace near the border of the sternum.

The regular heart sounds

The regular heart sounds are usually represented by the syllables, "lubb-dupp." The first sound is normally duller, louder and longer than the second; and the first sound produced by the left ventricle is duller and longer than that produced by the right. The first sound is composed of a valvular and a muscular element. The dulness and length of the sound depend on the strength and duration of the muscular contraction. In a strongly acting hypertrophied heart the muscular element predominates. The second sound is much shorter than the first, and is produced almost entirely by the closure of the semilunar valves, although it contains an element produced by the tension in the aorta and pulmonary artery. The pulmonary second sound, under normal conditions, is louder than the aortic in early life, but in middle life the sounds become equalized in intensity, and in later life the aortic sound is apt to be the louder. The first sound is normally louder than the second at the apex of the heart, and the second louder than the first at the base.

The time occupied by the heart sounds varies with the rapidity of the heart's action, but under normal conditions the relative intervals between these sounds are preserved within wide limits of variation in rapidity.

Diagnostic significance of variations from the normal in the regular heart sounds.

The following variations in *quality* may be observed: In children the valvular element normally predominates over the muscular element in producing the first sound, which is consequently shorter and more snapping than in the adult; and this same quality is often observed in thin, nervous adults. A sharp, snapping quality of the first sound, with diminution in its length, is also characteristic of weakening and perhaps thinning of the ventricular walls. This quality is found in cases with marked debility, as in chronic phthisis; also in anemia, in neurasthenia, and particularly in mitral stenosis, where it is a diagnostic sign of considerable value. The first sound is frequently not clear, but rather loud and rumbling and prolonged in robust adults with strongly acting hearts. In indolent people and those with fat chest walls it is apt to be short and somewhat indistinct.

The second sound may lose its clearness and show a dull, booming or flapping quality in old people with a stiffened or dilated aortic arch; and this quality may sometimes be observed in cases in which the action of the left ventricle is simply weakened.

A metallic quality of both sounds is observed in pneumopericardium, pneumothorax, or inflation of the stomach; and sometimes in cases with a large pulmonary cavity near the heart.

Impurity or roughening of the heart sounds may occur in healthy people, but it is often due to inflammation of stiffening of the valves not sufficient to produce a murmur; and it may be due to myocardial degeneration.

The *intensity* of the heart sounds depends not only on their character as developed by the heart, but also on the thickness of the chest wall and the presence of unusual tissues between the heart and the chest wall. It is also modified by position, being less in the recumbent than in the upright posture. In estimating the intensity of the heart sounds these modifying factors must be taken into account.

Increase in intensity of both sounds results from overaction of the heart produced by any cause; it is also observed when the heart is more than usually uncovered of lung or overlapped by consolidated lung; when cavities in the lung are situated near it, when there is pneumothorax; and when extra-pericardial adhesions exist.

Diminution in intensity of both heart sounds may be due to thick chest walls or interposed structures, or to feeble action of the heart.

The individual heart sounds may show modifications in intensity, as follows:

The first sound may be increased in intensity in both its muscular and valvular elements or in its valvular element alone. The former condition, observed at the apex, is most commonly due to hypertrophy of the left ventricle, in which case the second aortic sound is likewise intensified. Increased intensity of the valvular element of the first sound alone, observed at the apex, is noticed in cases of moderate dilatation and weakness in hearts previously hypertrophied; and not only in hearts which are the seat of pathological hypertrophy, but in those of athletes who have gone out of training. It is also observed in neurotic hearts and at the onset of acute febrile diseases. It is most noticeable in mitral stenosis. Increased intensity in both the muscular and valvular elements, heard in the tricuspid area, points to obstruction in the pulmonic circulation due either to lung conditions or obstruction at the pulmonic orifice.

Diminished intensity of the first sound alone at the apex is characteristic of myocardial weakness due to degeneration or dilatation. In cases of extreme myocardial weakness the first sound at the apex may be almost or quite inaudible, and the click of the second sound heard there is apparently but not actually accentuated.

Increased intensity of the aortic second sound heard both at the base and apex, the first sound at the apex being normal, indicates high arterial pressure or hypertrophy of the left ventricle from any cause. In atheroma of the aorta with stiffening of the semilunar cusps and without stenosis or incompetence, and in aneurism and dilatation of the aorta, the second sound may be loud, distinct, and perhaps harsh or metallic.

Increased intensity of the pulmonary second sound shows hypertrophy or overaction of the right ventricle. It is a diagnostic sign of importance in mitral disease and disease of the lungs which offer resistance to the pulmonary circulation, notably, pneumonia, emphysema, and phthisis.

Diminished intensity of the aortic second sound is noticed in marked mitral stenosis, in aortic stenosis and in aortic incompetence.

Diminished intensity of the pulmonary second sound, especially after it has been previously increased, is an important sign of failure of the right ventricle and of tricuspid incompetence.

The *pitch* of the heart sounds is to be distinguished from their degree of intensity of loudness. Their pitch may be high or low irrespective of their loudness. The pitch of the pulmonary second sound is the most important one to be considered. The aortic is normally higher than the pulmonary second sound in pitch, and if the pitch of the latter equals or rises above that of the former, resistance in the pulmonary circulation is suggested. This sign appears before accentuation or increased loudness is manifest. A lowering of this pitch after previous elevation is likewise an early sign of weakening of the right ventricle.

Variations in *rhythm* of the heart sounds are produced in two ways, by alterations in the relative lengths of the two silences and by reduplication of the heart sounds. The normal rhythm, which is well preserved at varying rates of speed, is in triple time, that is, the two heart sounds follow each other so as to correspond with the first two beats of triple time in music, the time of the third beat being represented by a silence, thus: *lubb, dupp,* ———, or *one, two, silence.* The accent is on the lubb or the dupp, according to conditions in the heart or to whether the stethoscope is placed over the apex or the base.

If the interval between the first and second sounds is so altered relatively as to become equal to the interval between the second and first sounds, the pendulum or tick-tack rhythm appears, which is also called embryocardia from its resemblance to the rhythm of the fetal heart sounds. Embryocardia is usually associated with rapidity and feebleness of the heart sounds, and indicates myocardial weakness.

If the interval between the first and second sounds is shortened relatively to that between the second and first sounds, the rhythm appears to be in quadruple time, thus: *lubb, dupp,* ———, ———. This rhythm may occur in emotional and febrile conditions with low arterial tension and a rapidly acting heart, but more frequently it is due to weakness of the ventricle relative to the arterial resistance which it must overcome, causing the ventricle to imperfectly empty itself and the semilunar valves to close prematurely. The appearance of this rhythm is usually a bad prognostic sign. It may occur in diphtheria, presaging fatal heart weakness. It is observed in digitalis poisoning with increased arterial tension.

Reduplication appears as a splitting of the first, or the second, or, rarely, both heart sounds; or, with a longer interval between the redoubled sounds, as a gallop rhythm. Its usual cause is probably asynchronism in the closure of the corresponding valves in the two sides of the heart, which results from inequality in the resistance of the systemic and pulmonary arterial systems, or in the systolic force of the two ventricles; but it may be that the third sound in gallop rhythm is sometimes caused by an unusually forcible contraction of the auricle.

Of the attempts made by cardiologists to represent onomatopoetically the variations in the regular heart sounds referred to by the terms reduplication, splitting and gallop rhythm, the following are, perhaps, the most successful: *tlat-tat* and *tat-tatl* for split sounds, and *ta-ta-tat* and *lub-dub-da* for the gallop rhythm.

Splitting of the first sound, which is much less frequently observed than that of the second, is sometimes found in arteriosclerosis and nephritis with high arterial tension; in myocardial degeneration with dilatation of the ventricles, and in obstructive pulmonary diseases. It is also occasionally heard in adhesive pericarditis. It may occur without pathological significance.

Splitting of the second sound is frequently heard in mitral stenosis and mitral incompetence, and occasionally in emphysema, pulmonary tuberculosis, broncho-pneumonia, and dilatation of the right ventricle. It also occurs in arteriosclerosis and nephritis with high arterial tension. It is not uncommon in diseases of the brain and in nervous conditions.

There is a form of reduplication of the second sound heard only at the apex, the second sound at the base being only accentuated, which is not due to asynchronism in closure of the semilunar valves, but is supposed to be due to sudden tension of the mitral flaps produced by the impact of the blood in the distended left auricle coming from that chamber into the left ventricle at the beginning of diastole. This form of reduplication is most frequently observed in mitral stenosis, and often merges into a distinct presystolic murmur. It has sometimes been heard in aortic incompetence, and also in apparently normal hearts.

The gallop rhythm, in which the doubled sounds are separated sufficiently to make the extra one appear like a new sound placed midway between the second and first sound, usually indicates a serious degree of myocardial weakness, and the prognosis increases in gravity with the rapidity of the gallop. It is a danger signal in arteriosclerosis, chronic nephritis, hypertrophic dilatation with broken compensation in valvular disease, myocardial degeneration, and dilatation with pericardial adhesions. It is an early sign in cardiac paralysis due to diphtheria.

ENDOCARDIAL MURMURS

Besides the regular heart sounds, variously modified in intensity, quality, pitch and rhythm, we listen in the precordium and its neighborhood for other, adventitious sounds which may be associated with the cardiac movements. These adventitious sounds are divided into two groups, accordingly as they are produced within or without the heart.

Endocardial murmurs or bruits are produced by derangements of the anatomico-physiological adjustments of the heart which cause sonorous vibrations to develop in the blood stream. These vibrations are communicated to the cardiac tissues and thence conducted by appropriate media to the surface of the body, where they are heard by the examiner as noises. In order to appreciate the diagnostic significance of these noises, it is necessary to understand something of the manner of their production.

PHYSICS OF ENDOCARDIAL MURMURS

Fluid passing through a tube is thrown into sonorous vibrations by the operation of two factors, the rapidity of the flow add encroachments on the lumen of the tube which produce eddies or obstructing currents. These encroachments may take the form of constriction of the lumen of the tube, of excrescences from its inner surface, or of roughening of its inner surface. The sonorous vibrations are produced on both sides of the obstruction and are transmitted both with and against the stream. They are communicated to the confining walls of the tube, and thence conducted by appropriate media to other regions. The intensity of the sounds produced by these vibrations, as heard, depends chiefly on the following factors: The rapidity of the current, a certain degree being necessary in any condition to produce audible vibrations; the specific gravity of the fluid, that of low specific gravity being more easily thrown into vibrations than that of a higher specific gravity; the nature of the obstruction; the character and thickness of the walls of the confining tube, thin walls conveying vibrations better than thick ones; and the character of the media which conduct the vibrations to the listening ear. Their distribution depends on the relative sizes of the cavities on either side of the obstruction, the larger volume of vibrations arising in the larger cavity; on the direction of the flow, the vibrations being transmitted more readily with the stream than against it; and on the character and position of the conducting media.

Looking at the heart as a tube through which the blood passes under pressure at a considerable rate of speed, we can apply the foregoing principles of physics to the explanation of the production of endocardial murmurs. We find in the pathological heart that obstacles to the blood flow frequently exist, the most common of which is obstruction of the heart channel at one or more of the cardiac orifices to which are attached the great valves. The obstruction may be positive, negative or relative. Stenosis of a valve is a positive obstruction. Incompetency of a valve is a negative obstruction, for the physical conditions under which the regurgitant flow takes place through the incompetent valve are essentially the same as those under which the direct flow takes place in stenosis. A relative obstruction is seen when the cavity on one or both sides of a cardiac orifice becomes dilated relative to the orifice. The effect of this on the production of murmurs is logically the same as if the cavities remained constant while the orifice was contracted. Other obstructions to the heart stream are caused by atheromatous excrescences, roughened areas of the endocardium, attached membranous shreds, vegetations, etc.

Still applying physical principles to heart conditions, we find that sonorous vibrations develop in the heart cavities on both sides of the obstruction, being greater in quantity in the larger cavity; and that they are transmitted more readily with the stream than against it. We find, also, that the audibility of these vibrations and the regions of the body surface where they are best heard depends largely on the character and anatomical arrangement of the conducting media.

These physical principles suffice for the explanation of all endocardial murmurs, both those due to permanent deformities of the heart tube, and those which are called functional or accidental because they are not associated with discoverable post mortem lesions. Given an endocardial murmur, we know that it must have its cause in some disarrangement, either permanent or transient, of the cardiac channel which creates an obstacle in the heart stream.

HOW MURMURS ARE PRODUCED IN PARTICULAR VALVE LESIONS

In *aortic stenosis* the valvular orifice is constricted or otherwise encroached upon. Vibrations develop on both sides of the obstruction, being greater in the larger cavity, the left ventricle. But that chamber is mostly covered by the right ventricle and the lung, while the smaller cavity, the aorta, comes near the chest wall in the second right interspace at the edge of the sternum; and the direction of the blood stream is toward the aorta. These facts explain why the murmur in this condition is usually heard with greatest intensity in the aortic area. It is also heard, though with less intensity, over the apex, because there the left ventricle comes in apposition with the chest wall. The comparatively superficial position of the great arteries in the neck and the direction of the blood flow explain why the murmur is most markedly transmitted upward over the course of those vessels. A relative stenosis caused by dilatation of the aorta immediately in front of the valves gives rise to a murmur with the same general characteristics as that due to stenosis of the valvular orifice.

In *aortic incompetence* regurgitation of blood takes place which is usually not free, but obstructed by remnants of the damaged valve; or, if incompetence is due to dilatation of the aortic ring, by the entire, but separated, semilunar cusps. The physical conditions here are similar to those in aortic stenosis, except that the flow is directed toward the ventricle and the larger cavity. The direction of the blood current and the dilatation of the left ventricle, which in this condition is usually considerable and brings the anterior wall of that chamber in extensive apposition with the chest wall, cause the murmur to be heard at the apex in most cases; though the fact that the auriculo-ventricular ring lies immediately above the aortic valves and easily transmits vibrations to the sternum explains why it is usually heard with maximum intensity over the sternum between the third and fifth costo-sternal articulations. As the aorta comes near the surface in the second right interspace near the sternum, this murmur is usually heard also, though with less intensity, in the aortic area, and may be transmitted some distance upward in the great arteries against the stream.

In *mitral stenosis* constriction at the left auriculo-ventricular orifice causes obstruction to the flow from the auricle to the ventricle. The auricle is usually well covered by lung, while the ventricle comes into contact with the chest wall at the apex; and the direction of the flow is toward the apex. Consequently the murmur is best heard at or just inside the apex. As the flow that produces this murmur is chiefly caused by the contraction of the comparatively weak auricle, the murmur is rarely loud or widely transmitted; although exceptionally it may be loud enough to be heard over the entire left ventricle and transmitted as far as the axilla. It is the most variable and inconstant of organic heart murmurs. A murmur similar to that produced by mitral stenosis, which is called the Flint murmur, is sometimes heard in cases of aortic incompetence which at the autopsy show no lesions of the aortic valves. In such cases the murmur may be produced by a relative stenosis due to great dilatation of the ventricle, or by a transient stenosis which results from the pushing forward of the mitral flaps in the dilated ventricle at the same time that the auricular contraction takes place, or the vibration of the mitral curtain caught between the two streams entering the left ventricle simultaneously from the left auricle and the aorta.

In *mitral incompetence* the valve flaps fail to coapt in the ventricular systole. This failure may be due to permanent deformity of the valve caused by organic disease, to dilatation of the auriculo-ventricular ring which prevents the valve flaps from coapting, to muscular weakness which prevents the auriculo-ventricular ring from contracting sufficiently to bring the flaps together, to weakness of the papillary muscles which prevents the cordeæ tendineæ from holding the flaps in firm apposition and allows them to bend backward into the auricle under systolic pressure, or to a lack of coordination in the complex muscular act by which the blood is expelled from the ventricle, which results in failure of the flaps to coapt at the right moment. The deformed or non-coapting valve flaps constitute an obstruction to the regurgitating blood stream, and the condition presented is essentially the same as in mitral stenosis, except that the direction of the current is reversed. Vibrations arise in both cavities, but are most numerous in the larger cavity, the ventricle. Moreover, the ventricle at the apex comes into apposition with the chest wall, while the auricle is usually well covered by intervening tissues. These facts explain why the murmur in the majority of cases is conveyed most strongly against the stream toward the apex and thence to the chest wall. Sometimes, however, when the auricle is much dilated or for other anatomical reasons lies in more extensive apposition with the chest wall than usual, the point of maximum intensity is over the auricle, in the second left interspace near the sternum. Ordinarily the vibrations which arise in the auricle are best heard in the back, between the inner edge of the left scapula and the spine at the level of the fifth or sixth dorsal vertebra. From the apex the murmur is conducted by the ribs toward the axilla.

In *pulmonary stenosis* the murmur is produced in the same way as in aortic stenosis and is heard most distinctly over the smaller chamber, the pulmonary artery, which comes close to the chest wall in the second left interspace near the sternum. The murmur is transmitted a short distance upward and to the left, and is then carried into the interior of the chest, where it is lost or heard in the back on both sides of the spine between the scapulæ. The vibrations in the right ventricle are usually heard over the lower end of the sternum. If the murmur is a loud one, which is often the case, it may be audible over the entire chest.

In *pulmonary incompetence* the murmur is heard with greatest intensity over the pulmonary artery, where it approaches the chest wall in the second left interspace near the sternum, and is carried by the blood stream downward into the right ventricle, and is audible along the left border of the sternum as far as the fourth interspace.

In *tricuspid stenosis* the development of the vibrations and their conduction to the surface take place in the same manner as in the corresponding lesion of the left heart, and the murmur is transmitted to a limited area near the lower end of the sternum.

In *tricuspid incompetence,* as in mitral incompetence, the vibrations are most strongly developed in the larger chamber, the ventricle, and are carried against the stream to the tricuspid area, at the lower end of the sternum. This murmur usually is not far transmitted, although when the right auricle is much dilated it may be heard as far as the right nipple line. It is not regularly heard in the back, as is the case in mitral stenosis, for the right auricle does not, like the left auricle, when dilated, come extensively into apposition with the posterior chest wall.

The anatomical derangements which give rise to the so-called *functional murmurs* cannot be clearly identified, because they are not discoverable in the autopsy. But it is possible to conceive of conditions capable of producing them. Muscular weakness or incoordination, which prevents the auriculo-ventricular ring from contracting sufficiently to bring the mitral flaps together, or a similar weakness or incoordination which prevents the papillary muscles from holding firm the cordæ tendineæ to which the mitral flaps are attached, may produce a transient incompetence of the mitral valve and give rise to a murmur identical with that produced by an organic mitral incompetence. Dilatation of the pulmonary artery directly in front of the semilunar valves due to relaxation of its walls, and dilatation of the conus arteriosus of the right ventricle, both present conditions of relative stenosis of the pulmonary orifice capable of producing a characteristic murmur in the pulmonary area, where most hemic murmurs are heard. In the existing state of our pathological knowledge it is possible only to speculate on the changes in the heart which produce these functional murmurs, but if we can exclude exocardial murmurs, such as those produced by the heart on small portions of pinched lung, and those arising in the great veins of the neck which are transmitted to the vena innominata or vena cava, we must infer that functional murmurs are produced by actual though transient changes in the shape of the heart channel sufficient to produce audible vibrations, which may or may not be associated with increased speed of the cardiac stream in the expulsion period, or with diminished viscosity of the blood.

DIAGNOSTIC SIGNIFICANCE OF ENDOCARDIAL MURMURS

The fact that an endocardial murmur is heard points to an obstruction either absolute or relative in the heart channel, but it does not tell us the nature or the location of the obstruction. In order to get the full diagnostic significance of such a murmur we must note the following additional facts about it: The spot where it is heard most distinctly, its area of audibility and the directions in which it is most strongly transmitted, its place in the cardiac cycle, its intensity, its quality, its persistency, the effect on it of exertion, respiration and position, the presence of thrills, changes in the regular heart sounds, changes in the size and shape of the heart, changes in the circulation, coexisting diseases and symptoms, the previous history of the patient, and the relative frequency of the different lesions producing it.

The *points of maximum intensity* of murmurs referable to particular valve lesions lie in the areas in which the regular closing sounds of the valves are best heard, with some exceptions. The point of maximum intensity of the murmur of aortic incompetence is less commonly observed in the aortic region than elsewhere, being more frequently found in the midsternal region, or in the third left interspace, or at the apex; and the murmur of mitral incompetence may be heard most distinctly in the pulmonary area when dilatation of the left auricle or retraction of the lung brings that chamber into unusually extensive apposition with the chest wall.

The *area of audibility and directions of transmission* of murmurs vary greatly, some being heard only in very limited areas, while others are heard over wide regions of the chest and even at some distance from the chest wall. The murmurs of mitral and tricuspid stenosis are regularly heard only within small areas, while those of aortic and pulmonary stenosis are often widely audible. But even when the area of audibility is extensive it is usually possible to make out the characteristic directions in which the murmur is most strongly transmitted.

The murmur of aortic stenosis is most markedly transmitted upward, over the carotid and subclavian arteries. It is also usually transmitted downward to the apex.

The murmur of aortic incompetence is most markedly transmitted downward to the base of the sternum and to the apex. It is also usually transmitted upward for a short distance.

The murmur of mitral stenosis is rarely transmitted far in any direction, but occasionally it may be heard as far as the midsternum, and when the apex comes into unusually close apposition with the chest wall it may be transmitted for a few inches toward the axilla.

The murmur of mitral incompetence is usually transmitted toward the axilla, and also toward the sternum and upward when the left auricle is dilated and uncovered of lung. It is frequently transmitted to the back between the left scapula and the spine at the level of the fifth rib.

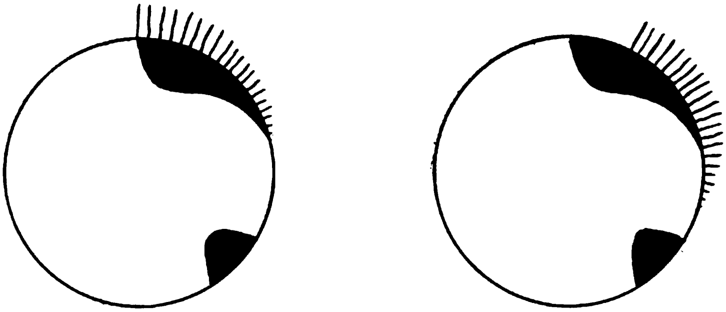
The murmur of pulmonary stenosis is transmitted slightly upward and to the left, and also to the back, between the scapulæ, on either side of the spine.

The murmur of pulmonary incompetence is transmitted downward along the left edge of the sternum.

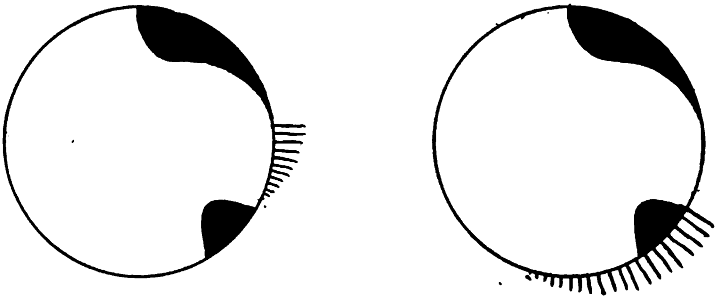
The murmur of tricuspid stenosis is rarely transmitted beyond the tricuspid area at the base of the sternum.

The murmur of tricuspid incompetence is usually transmitted upward and to the right for a short distance.

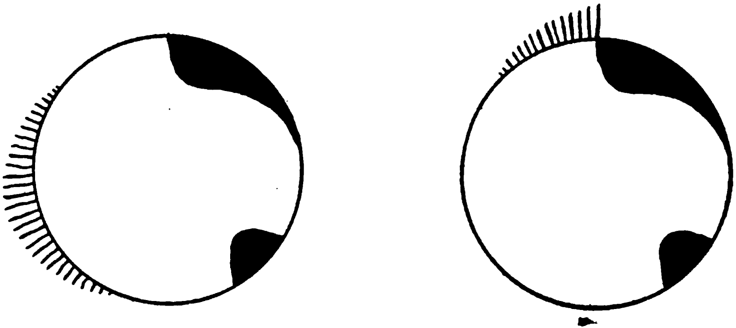
To determine the *place in the cardiac cycle* to which a murmur belongs is of the first importance in diagnosis. The cardiac cycle can be identified by the two regular heart sounds, and the murmur should be placed in its relation to those sounds. If a murmur is systolic we know that it can be caused only by stenosis at the aortic or pulmonary, or incompetence at the mitral or tricuspid orifices. The systolic murmurs of aortic and pulmonary stenosis differ slightly in phase from those of mitral and tricuspid incompetence; they begin later, at the end of the closure time of the auriculo-ventricular valves, when the pressure inside the ventricles equals that in the aortic and pulmonary arteries; while systolic mitral and tricuspid murmurs usually begin with the contraction of the ventricles and more or less completely replace the first sound. There is a form of systolic murmur due to mitral incompetence as sometimes observed which is heard late in the systole, but before



SYSTOLIC MURMUR LATE SYSTOLIC MURMUR



PRE DIASTOLIC MURMUR DIASTOLIC MURMUR



MID DIASTOLIC MURMUR PRE SYSTOLIC MURMUR.

Diagram showing the relation of endocardial murmurs to the regular heart sounds the diastole, and is called the prediastolic murmur; it is probably caused by the mitral flaps, after they have once closed, bending backward into the auricle under the full expulsion pressure of the ventricular systole. This murmur may indicate a slight organic valve lesion or weakness of the auriculo-ventricular ring or the papillary muscles. Murmurs heard at the beginning or throughout the diastole may mean incompetence of the aortic or pulmonary or stenosis of the mitral or tricuspid valves; but those heard only in the middle or latter part of the diastole point exclusively to mitral or tricuspid stenosis. The murmur of aortic incompetence is the most constant of murmurs, and that of mitral stenosis is the most variable. The latter may occupy the entire diastole or any part of it, though it is most commonly heard only in the latter portion, when it is called presystolic. It is often absent.

The *intensity or loudness* of murmurs possesses, per se, only a moderate amount of significance in the diagnosis of valvular lesions, because it depends as much on the action of the heart as on the nature of the lesions. With fairly good heart action, organic murmurs are usually louder than those due to functional disturbance, and systolic murmurs are in general louder than diastolic ones. In stenosis the murmur may increase in intensity with increase in the stenosis up to a certain limit, but in incompetence an increase in the amount of regurgitation is usually attended with diminished intensity of the murmur, and an extreme degree of incompetence may show a very faint murmur. Variations in the loudness of organic murmurs are often important indications of increase or diminution in the strength of the heart action. Increased loudness of a murmur which is faint when the heart action is feeble is usually a good sign.

The *quality* of murmurs is variously described as harsh, rough, rasping, musical, purring, rumbling, blubbering, swishing, blowing, etc. The diagnostic significance of these differences in quality is not very great. In general, it may be said that organic are usually harsher and rougher than functional murmurs, and that those due to stenosis are apt to be harsh, rough or musical, while those due to incompetence are more often soft and blowing. Only one murmur, the mitral or tricuspid presystolic, is purely crescendo.

The *persistence* of a murmur bears directly on the diagnosis, for if it persists it usually means an organic valve lesion or a permanent dilatation of a valvular orifice; while if it disappears under equal conditions of heart action, it is probably functional or produced by a transient dilatation or other accidental cause.

The *effect of exertion, respiration and position* on murmurs may be briefly stated as follows: Exercise usually intensifies feeble murmurs due to slight or compensated lesions. If, however, a period of rest makes a murmur more distinct, the lesion is apt to be a serious one. Organic murmurs are best heard at the end of respiration, when the heart is most uncovered of lung. Functional murmurs are sometimes best heard at the close of inspiration. The recumbent position increases the distinctness of functional murmurs and those due to mitral and tricuspid incompetence, diminishes the distinctness of those due to mitral stenosis, and does not affect the distinctness of those due to aortic stenosis and incompetence.

Thrills are important corroborative signs in the diagnosis of valvular lesions. Felt over a valve area in which is heard a characteristic murmur they usually confirm the diagnosis of an organic lesion of that valve. They have already been described in the section on palpation.

The diagnostic significance of *changes in the regular heart sounds* observed in connection with murmurs is positive and considerable. The closure sounds of affected valves may be replaced in whole or in part by murmurs, and the closure sounds of other valves may be altered. If the closure sound of a particular valve is completely replaced by a murmur referable to that valvular orifice, the lesion is apt to be more extensive than if it is not completely replaced. If the pulmonary second sound is strongly accentuated in the presence of a murmur referable to the mitral orifice, it indicates compensatory hypertrophy of the right ventricle and supports the diagnosis of an organic mitral lesion. Weakness of this sound after a previous intensification, points to failure of the compensatory hypertrophy and secondary incompetence of the tricuspid valve. In the presence of a murmur suggestive of tricuspid incompetence weakness of this sound is a valuable confirmatory sign. The aortic second sound is usually weakened in mitral stenosis and often in mitral incompetence when imperfect compensation exists.

Any condition which diminishes the quantity of blood thrown into the aorta by the ventricular systole weakens this sound. In mitral stenosis the first sound is apt to have a short, flapping character. In aortic stenosis and incompetence with good compensation, the first sound is apt to have a dull, booming quality due to hypertrophy of the left ventricle. If dilatation prevails over hypertrophy, the first sound is apt to be short, high pitched and flapping in character, as well as faint.

Enlargement of the heart observed in connection with murmurs, is a diagnostic sign of the first importance. It almost always means an organic lesion; and a murmur which is not accompanied by a demonstrable alteration in the size or shape of the heart is either functional or produced by a very moderate organic lesion which is compensated. Enlargement of the different heart chambers is regularly associated with lesions of particular valves, the amount of which depends on the character of the lesions and the degree to which compensation has been lost.

With aortic stenosis we usually find a moderate enlargement of the left ventricle, which in bad cases with ruptured compensation may be considerable. A much greater enlargement of this ventricle is commonly observed with aortic incompetence, which in bad cases may be so great as to carry the apex to the seventh interspace and the anterior axillary line. The right heart is not enlarged in aortic lesions unless there is also failure of the mitral valve.

With mitral stenosis the two chambers immediately behind the valve, the left auricle and right ventricle, are enlarged, and the right auricle also, if the compensatory hypertrophy of the right ventricle fails and the tricuspid valve gives way. With mitral incompetence there is dilatation of the left ventricle and auricle and the right ventricle, and of the right auricle also if the tricuspid valve gives way. The enlargement of the heart in mitral disease is chiefly transverse, while in aortic disease it is chiefly longitudinal. In extreme cases of mitral incompetence with ruptured compensation the dilatation may be so great that the area of cardiac dulness extends from the right mammillary to the left midaxillary line.

Lesions of the pulmonary valve produce enlargement of the right ventricle in the same way that lesions of the aortic valves produce enlargement of the left ventricle.

Tricuspid stenosis produces enlargement of the right auricle and tricuspid incompetence enlargement of both chambers of the right heart.

Changes in the circulation in the circulation observed in connection with murmurs sometimes help in their interpretation. The arterial pulse in pure aortic incompetence is often pathognomic, being rapid, with a powerful systolic wave which feels under the finger like the passage of a shot. In aortic stenosis it is apt to be hard, rather slow, with a small wave. In mitral stenosis it is unchanged or small, or, when compensation is lost, irregular. In mitral incompetence it shows no change unless compensation is lost, when it is irregular. In any case of valvular disease with great loss of compensation, it is feeble and irregular. Systolic pulsation of the veins in the neck, seen in connection with the murmur of tricuspid incompetence, is a pathognomonic sign of that lesion, as is also diffuse pulsation of the liver. Passive congestion of the lungs and abdominal viscera, and edema, particularly of the lower extremities, in the presence of a heart murmur, are confirmatory of valve disease with ruptured compensation.

Coexisting diseases and symptoms and the previous history of the patient may throw light on the meaning of heart murmurs. A murmur occurring in the course of rheumatic fever or scarlet fever, or in an old person with atheromatous arteries, points to an organic valvular lesion. If it occurs in the course of anemia or of a nervous disorder attended with rapid or violent heart action, it is more likely to be functional. If a person with a heart murmur suffers from dyspnea on slight exertion and a dry cough, an organic valve lesion may be suspected. If a person showing a heart murmur gives a history of having had a disease which commonly produces endocarditis, or of having had signs and symptoms indicative of heart failure, the diagnosis of organic valve disease suggested by the murmur is greatly strengthened.

Consideration of the *relative frequency of different valve lesions* sometimes helps in the interpretation of those murmurs which point to more than one lesion. A systolic murmur heard with greatest intensity in the pulmonary area is more apt to be due to anemia or mitral incompetence than to pulmonary stenosis, because the last is a rare condition, while the others are common. A diastolic murmur heard only in the midsternal region or down the left edge of the sternum is much more likely to be due to aortic than to pulmonary incompetence for the same reason. Similarly a presystolic murmur heard anywhere between the apex and the lower part of the sternum suggests more strongly a mitral than it does a tricuspid stenosis.

DIAGNOSTIC METHODS IN GLÉNARD'S DISEASE: ENTEROPTOSIÉ

By A. ERNEST GALLANT
Consulting Surgeon, Jamaica Hospital and the Eastern Long Island Hospital, Greenport
New York

Synonyms: Visceroptosis, splanchnoptosis, nephroptosis, floating —movable—prolapsed—displaced kidney, liver, stomach, etc., or applied to any viscus within the confines of the abdomino-thoracic cavities.

Thirty-one years have now passed since Francis Glénard published his epoch-making paper detailing an intimate study of forty-five patients suffering from what he so aptly named "Enteroptosié" and which has since been most justly entitled "Glénard's disease." Yet how few really know just what Glénard taught or wherein his observations outstepped those of his predecessors. Succinctly stated —(a) that while a prolapsed right kidney might be the cause of the most notable symptoms (Dietl's crises); the most easily located and the most readily replaced, the kidney only served as a "sign-post or index" pointing to the presence of a greater or lesser degree of prolapse of all the viscera; (b) describing a very simple, yet reliable means of making a diagnosis of "Visceroptosié" and (c) its intimate if not all important relation to mal-nutrition, and so-called neurasthenia and nervous prostration.

Unfortunately, Glénard's book has never been translated, and for this reason, chiefly, in spite of the many hundreds of articles and monographs which have been published on this subject, under Glénard's disease or some synonym (visceroptosis, enteroptosis, splanchnoptosis, or the name of an organ with 'ptosis added), how comparatively few practitioners make a positive diagnosis and direct their patient as to the best means of treatment.

In order to "lend a helping hand" we present a real "bakers dozen" of ways and means, whereby an unequivocal diagnosis of Glénard's disease can be determined.

When about to make a diagnosis it is always well to keep prominently in mind (a) that in but very few exceptions whenever one organ or viscus has prolapsed it is a sure indication that many if not all are to a greater or less extent prolapsed; (b) that in order to apply the proper treatment it is *not* essential to know just which organs or how many, or how far downward each is displaced; (c) nor need we trouble to determine just which symptoms are due to this organ or that viscus; but simply treat the general condition as a whole, and in good season, many otherwise inexplicable symptoms will vanish or their real origin be made manifest, and appropriate treatment can be instituted. This rule is especially true, in the relation to the genitourinary tract in females.

At birth the small abdominal cavity and shallow pelvis is occupied chiefly by the liver; the kidneys and spleen occupying the wings of the false pelvis.

As the child develops and the spinal bodies thicken, the solid organs are carried upward, and the lower abdomen is filled by the stomach and lengthening intestines, as seen in the "pot-belly" of childhood.

Between the tenth and fifteenth year the spine lengthens at twice the rate that it has before or after that period; the lumbar vertebrae seem to grow in excess of all the others and this, coupled with the deepening and broadening of the pelvis increases the abdominal capacity, to a marked degree, and the tendency to splanchnoptosia is directly in proportion to the length of the spine, the length of the trunk and the distance from the pelvic floor to the diaphragm, or as we have found most practical for record, the length from the top of the symphysis pubis to the supra-sternal notch. Practically in every man, woman and adolescent child in whom this measurement is 19 inches or over we find more or less abnormal mobility downward of all the viscera of the pelvis, abdomen and thorax.

While it is undoubtedly true that this condition may be brought about by the hop-skip-and jumping of childhood, the severe cough of la grippe, excessive indulgence in tennis, and rope jumping contests at just the growing age, and that the kidneys may be traumatically displaced by falls, blows, strains, etc., yet the one *all important underlying cause effecting nearly one-half of all creation,* is not relaxation of the abdominal wall (this comes later) but because the foundation—the pelvic floor—*is too far below the diaphragm* or in other words the space below the diaphragm is too capacious and the inconstant, ever varying contents of the alimentary tract too unstable to afford a buffer which will withstand the jolts and jars to which these long-waisted folk are subject.

Methods for making a diagnosis of Glénard's Disease (any one of which will "tell the story").

Method 1. *Glénard's test.*—"Stand behind the patient, reach your arms around her body, interlock the fingers over the suprapubic area, thus embracing the whole lower abdomen, then lift upward and backward. This affords the patient a grateful sense of support and comfort which is followed by a sensation of 'everything falling' when the examiner suddenly releases his hands."

Method 2. *The Facial Expression* presents a characteristic index —(a) a flattening of the nasal portion of the cheek; (b) a hollowing under the eyes; (c) an unnaturally deep fold running to the angle of the mouth; in all a lack of that fullness or roundness of the cheek which mars any human face.

Method 3. *The Voice* in ordinary conversation is usually of good quality and pitch, but when repeating for the 'steenth time the story of her ills, aches and ailments, she runs over the details in a sing-song, whining tone, aiming to elicit that sympathy and interest which has failed her at the hands of the five, ten or twenty physicians whom she has before consulted.

Method 4. *The Gait.*—When walking we note the quick, nervous, indecisive step, the early fatigue, on exertion, excepting when dancing, in which exercise they will tire to exhaustion, several well set-up men. The association, excitement and music seems to develop an extraordinary degree of endurance; followed by a severe fatigue and reaction which will keep them in bed until near the noon of the next day.

Method 5. *The Figure.*—Two types are met with (a) the underweighted, undernourished individual, round-shouldered, flat-chested, more or less "pot-bellied," when standing, and doubled up when seated, showing a total lack of the muscular tone necessary to maintain an upright attitude, really a condition of imbalance *due to the change in the location of the organs,* and the involuntary effort to counter-balance the weight center by throwing the shoulders back and the abdomen forward. (b) In those of the second or "aldermanic" type the excessive accumulation of soft, flabby fat, especially over the lower abdomen, exaggerates the ungainly attitude, so characteristic of the overfed citizen who tires so readily on slight exertion that he makes matters worse by refraining from any effort or exercise which can be avoided.

Method 6. *The Abdominal Outline.*—Naturally, the stomach occupies the epigastrium and causes the abdominal wall to bulge, sloping down gradually to the symphysis pubis. In visceroptotic patients the reverse of this is present—the stomach now occupies the *hypo*gastrium, which it causes to protrude and the *epi*gastrium is flattened or actually hollowed, a sunken-in "crater" just below the ensiform; in profile an outline which we have compared to an inverted interrogation mark and always associated with ptosis or dilatation of the stomach and transverse colon.

In many thin men, girls and young nuliparae when standing the suprapubic prominence does not exceed one-half inch, and when lying down the hypogastric area seems to actually recede, leading the patient to remark, "I never had a high stomach," which is really true, but the presence of the gastric resonance in the hypogastrium will confirm the diagnosis.

For twenty years we have made use of the orthopedist's lead tape for determining and recording the abdominal outline.

As pointed out by Mark Knapp, in many thin or undernourished patients, some when standing, others when reclining, the contour of the stomach, colon and even the small intestines and their peristaltic waves become visible through the abdominal wall. The abdominal palpitation in the epigastric and umbilical regions, so often a source of worry, is simply caused by the aortic pulsation, transmitted through the lax abdominal wall.

Method 7. *The Long Waist.*—Anthropologically and etiologically speaking a long trunk and Glénard's disease go hand in hand,

and a long experience justifies the following statements—(a) That visceroptosia is found in every long waisted (long trunked) man, woman and child; (b) that all those whose pegulo-pubic measurement (from the top of the symphysis pubis to the supra-sternal notch) is 19 inches or over, independent of the length of the lower extremities, must be classed as "long-waisted." Occasionally we meet with visceroptosia in women whose pegulo-pubic distance is but 18.5 inches, and in women who have borne several children, and have not been provided with proper and efficient abdominal support during and after the puerperium.

N. B.—The pegulo-pubic measurement must be taken with the patient lying flat on the back.

Method 8. *Palpation* of the abdominal viscera through the abdominal wall excepting perhaps the kidney and liver, to be of *determining* value must be the result of constant practice in abdominal manipulation to enable one to apprehend the appendix, track the colon, catch the kidney, lift the liver, grasp the gall-bladder, etc. When palpating for prolapsed kidney some succeed best when the patient stands, leaning forward, the hands resting on the back of a chair (Goelet), or lying flat on the back (Edebohls), or in the left lateral posture (Martin); but in our experience the most satisfactory position is when lying on a firm couch in a semi-reclining attitude, when the kidney naturally drops and the abdominal wall is relaxed to the greatest degree.

To secure complete abdominal relaxation in some patients it is necessary to tell them to breathe through the mouth; and in others to keep them answering irrelevant questions.

The light passage of the hands may cause an intolerable tickling, or a certain amount of soreness and even pain, which greatly interferes with the examination, but can usually be overcome by increasing the degree of pressure. Every student of abdominal palpation should practice this on his own abdomen or permit an associate to do so, as the only reliable means of learning what this statement means.

One must never forget that (a) thoracic breathing is the rule with women; that (b) many cannot take an abdominal breath, that (c) when told to take a deep breath they lift the diaphragm and the kidney upward out of the reach of the examiner's hands; and it is only when instructed to let all the breath out, in expiration, that the displaced kidney can be palpated. This maneuver may have to be repeated in order to make a positive diagnosis.

Succussion.—The "Splashing" sound elicited by sharply compressing and quickly relaxing the abdominal wall over a stomach partly filled with fluid, is usually considered a positive evidence of stomach dilatation with or without ptosis.

Method 9. *Percussion.*—With the fingers of the left hand spread apart and firmly applied over the epigastrium and striking one finger after the other from above downward the low-pitched, hollow, drumlike stomach-note can be readily recognized, and differentiated from the dull key when the upper border of the stomach is absent and dragged downward. By the spreading fingers of the left hand below the umbilicus the lower limit of stomach resonance can be readily determined. Of course the ear must be trained in order to discriminate all the other viscera.

Method 10. *Illumination.*—Transillumination of the stomach by the gastrodiaphane of Einhorn, is a very graphic means of convincing the patient of the reality of her condition, and if the cable is long enough and the greater curvature within the true pelvis it is possible to see the light through the vaginal speculum.

(b) The gastroscope of more recent date affords a means of inspecting and locating foreign bodies and ulcers, etc., in the stomach and with (c) the fluoroscopic screen one may even watch the bismuth filled gastrointestinal tract in action; but of course each and all of these have a limited field of usefulness, and have been superseded by the invaluable discovery of Röntgen, so that with the newer tubes, tables, etc., in the hands of competent men all the hidden mysteries of the human anatomy are made visible and can be recorded on the photographic plates, and reproduced in the "movies."

Method 10. *Statistically speaking* perhaps the most important contribution to this subject is that of Levy and Kantor, on "The Incidence of Visceroptosis," the result of a study of 1,600 patients who applied for treatment for "Stomach trouble." Of these 898 were X-rayed and there was found present marked gastroptosis in 46 per cent.; more or less gastroptosis in 64 per cent.; with an associated ptosis of the liver, 70 per cent.; right kidney, 30 per cent.; both kidneys, 3 per cent.; the spleen, 1 to 2 per cent.

Their observations as to sex (5 men to 6 women) was a revelation and upsets most of our former statistics.

Method 11. *Postural Diagnosis.*—Having palpated, percussed, successed (perhaps otherwise "cussed") and marked out on the abdominal wall the location of the several viscera, hollow and solid, our findings may be confirmed by putting the patient in bed with the foot elevated ten inches, to sleep overnight. In the morning, after massering the abdominal contents upward toward the diaphragm, with a pen or pencil of another color map out the changes in their position brought about by the elevation. Occasionally adhesions within the abdomen will prevent one or several viscera from receding toward the diaphragm, but this will only confirm the diagnosis of prolapse, as will also the improved heart's action, the slowing and deepening of respiration after a day or two and the filling of the hollows in the cheeks, especially if the patient drinks a gallon of water per day.

Method 12. *Symptomatically* we divide these cases into (a) acute, (b) recurrent; and (c) chronic. Briefly, acute attacks may be due to traumatic displacement of the right or left kidney, with nausea, vomiting, pain and swelling along the right loin, with or without kidney blood in the urine. These symptoms continue until the patient is put in bed and the kidney slips back into its nest. This syndrome has been recognized and spoken of as "Dietl's crises."

(b) *Dietl's crises* may recur from time to time, whenever the patient sits up or after an interval of days, weeks or months, and the diagnosis can be cleared up by placing the patient in bed with foot elevated.

Jaundice, as we have elsewhere pointed out, is not only a very frequent index of visceral displacement, but our experience leads to the belief that ***the genesis of jaundice is always due to traction of a prolapsed kidney on the cystic or common duct.*** The elevated posture always clears up the jaundice.

(c) *Chronic symptoms.*—It is as from this group that as a profession we derive our chief income—including as it does the neurasthenic, the social wrecks and business failures with nervous prostration or borderline insanity, all the pessimists who weigh us down on this terrestrial globe, and prevent our enjoyment of the Swedenborgian heaven, and delay the arrival of the true millennium.

These are the patients who require of us the practice of the greatest patience, and demand our most patient consideration, and when properly handled respond most readily, and appreciate our efforts most fully.

Chronic enteroptotics always complain of "stomach troubles" and intestinal sluggishness, they either over-eat or fear to eat so many things as to really suffer from want of sufficient nourishing food (sitophobia, Einhorn) and usually are from 10, 20, 30 or more pounds underweight; or else carry a similar amount of overweight, of flabby fat which not only impedes the heart's action but increases the difficulty of amply aerating the lungs within their flattened chest walls.

Appendicular constipation, obstipation, and obstruction (so-called chronic or recurrent) causing colicky attacks, which reflexly upset the stomach, as evidenced by the gastric unrest, nausea, vomiting, intermittent pain at McBurney point, discomfort on walking, necessitating the flexing of the thigh on the abdomen especially at night, frequently accompanied with high fever, and leukocytosis, with sudden cessation of the pain, etc., when either the offending enterolith is forced past the obstructing bend, kink, twist, etc., and escapes into the caput coli and for the time being ends the danger, or failing this, rupture of the appendix takes place and we then have to deal with a perforated appendix. The surgeon who at this stage can positively determine which of these processes he has to deal with (emptying or perforation?) will be able to attain a *zero* mortality; but failing in this infallibility, when in doubt we operate.

Nervous and mental instability is always a very prominent feature, so much so that many will have consulted "nerve specialists," osteopaths, chiropractics, etc., and failing in the "faith" which can alone make them whole "and wholly Christian Scientists, they drift from one doctor to another, from regular to 'pathy, seeking rest and cure, but finding none," they too often try to appease the demon of unrest in Turko-tango-trotting and under the stimulus of cigarettes, alcohol, and cabarét they dance the hours away, returning home in a condition of irritability, nervousness and restlessness, and in order to sleep resort to some sort of "dope," adding fuel to the fire which is burning up their health. How much coincident acidosis and intestinal toxemia secondary to the 'ptoses have to do with this nervous unrest it is difficult to estimate. In many cases hypothyroidism seems to play an important part; hyperthyroidism certainly exaggerates all the symptoms, and should never be overlooked.

Painful, irregular, scanty or excessive menstruation is another source of irritation and when accompanied with headache frequently "drives her almost distracted," to drink or worse, for relief.

Unfortunately the coincident *cardioptosis* of Glénard's disease has received but too little attention and it was with pleasure that recently we listened to a paper read by Charles Lyman Greene (to be published) on the "Prophylaxis and retardative treatment of cardiovascular disease" in which he called attention to the relative frequency of "Dropped heart" and by many X-ray plates showed to what extent the apex may be dislocated, even to a vertical position, and the aorta and its vessels be distorted.

Another neglected phase is the relation of the diminished chest capacity and flattening due to traction, with the lessening of the respiratory expansion and its influence as a predisposing factor in tuberculous infection?

Method 13. *Diagnostic therapeutics.*—After all the most satisfactory "method" is that whereby our diagnostic "guesses" are transformed by diagnostic therapeutics into curative successes. The means by which in our hands this has been achieved may be summarized as follows:

(a) The patient must be made to understand that there is a very real physical basis underlying the condition, which must and can be overcome.

(b) That by following the prescribed régime in every detail she can be restored to health and strength.

(c) The family and friends must keep "hands off" absolutely.

(d) Rest, mental and physical, with isolation.

(e) Postural replacement.

(f) Reparative operations on nose, throat, pelvis, etc.

(g) Recuperative massage, active and passive movements.

(h) Efficient abdominal support, by plaster, "stock binder," corset.

(i) Medicinal tonics.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

Amylase in the Blood Serum and Urine—P. STOCHS, Quart. Jour. Med. (London), April, 1916.

Amylolytic ferment is present in the blood serum and urine of all healthy individuals and has also been found in all the body fluids examined. The level is practically constant in the blood serum—the level in the urine is subject to diurnal variations due chiefly to the digestive functions. The ferment is of pancreatic origin and is absorbed directly by the blood. No proof of the action of antiamylase has been found. Disease of the kidneys causing any diminished permeability of these organs reduces the amount of ferment in the urine and consequently causes the amount in the blood to rise. Any disturbance in the ratio D (blood): D and M (urine) indicates renal insufficiency in all such cases. Severe passive congestion also raises the amount of amylase in the blood. With these exceptions, any increase of the ferment in the blood serum denotes pancreatic mischief. The values have been found raised in all cases of pancreatic disease, the increase depending on the degree of obstruction in any part of the gland or its ducts and on the acuteness of the condition. The highest values were found in cases of acute pancreatitis. The estimation of the amylolytic capacity of the blood serum and the urine is a most delicate test of the efficiency of the pancreas, and consequently is a most delicate and reliable test for disease of the pancreas. The use of the simplified modification of Wohlgemuth's method and the use of an identical technic for serum and urine are justified by the consistency and regularity of the results obtained.

SACHS.

Ninhydrin Color Reactions of Proteins and Their Split Products—H. W. EMERSON and J. S. CHAMBERS, Jour. Lab. and Clin. Med., July, 1916.

Vaughan's protein poisons in dilutions up to (1-10,000) give the ninhydrin reaction. The proteins from which the poisons are obtained will also give the ninhydrin reactions in dilutions up to (1-10,000). The cell residue in dilutions of (1-100) does not give the ninhydrin reaction. Dilute acids and alkalies interfere with this reaction. Sodium chlorid interferes very slightly with the reaction.

Prolonging the time of heating makes the reaction very much more delicate. WESTERN.

Non-protein Nitrogenous Constituents of Blood and Phenolsulphonephthalein Test in Children—J. S. LEOPOLD and A. BERNHARD, Am. Jour. Dis. Child., June, 1916.

Figures for the non-protein constituents of the blood as well as for the phenolsulphonephthalein excretion of children free from renal disease are practically identical with the figures obtained from adults, and vary within the normal limits as the adult figures vary. The importance of the tests for diagnosis and prognosis amply demonstrated in adults, will, in all probability, hold true for children, although a more comprehensive series of cases are required definitely to establish this view. SACHS.

Amino-Acid Minimum for Maintenance and Growth—F. B. OSBORN and L. B. MENDEL, Jour. Biol. Chem., May, 1916.

Osborn and Mendel report additional experiments showing the importance and the part played by tryptophan and lysin in the metabolism and maintenance of growth. The quantity of these amino-acids in the diet can be made the limiting factors in determining the nutritive equilibrium and the possibility for increase in size of the individual. SACHS.

Effects of Surgical Procedures on Blood-Sugar Content—A. A. EPSTEIN and P. W. ASHNER, Jour. Biolog. Chem., May, 1916.

In 12 cases in which a hyperglycemia (sugar content above 0.11 per cent.) was found before operation, it was established that no psychic disturbances were accountable for the condition. The hyperglycemia in these cases was due to various factors such as obesity, nephritis, hypertension, fever and loss of blood. In 57 out of 60 patients operated on under general anesthesia, there was an increase of the blood-sugar content. Authors state that the anesthesia plays an important part in the production of the hyperglycemia. SACHS.

Clinical Observations on the Pineal Gland—G. HORRAX, Arch. Int. Med., May, 1916.

Author studied 3 cases of supposed pineal tumor which demonstrated that tumors of the pineal gland in children, occurring before the age of puberty, usually give rise to a syndrome characterized by precocious development. In one case, a boy of 12 years of age, there was a precocious adolescence and intra-cranial symptoms. Autopsy revealed struma of the pineal gland.
SACHS.

Experimental Observations on the Pineal Gland—G. HORRAX, Arch. Int. Med., May, 1916.

Author states that pinealectomized male guinea-pigs show a hastened development of the sexual organs. The pinealectomized female guinea-pigs tend to breed earlier than controls of the same age and weight. SACHS.

Myopathy and Disorders of Internal Secretions—G. P. McCOUCH and S. D. W. LUDLUM, Med. Rec., June 10, 1916.

Myotonia congenita and myasthenia gravis are frequently associated with muscular dystrophy. Amyotonia congenita has occurred in connection with muscular dystrophy and may be identical with one form of it. There is some evidence suggesting that thyroparathyroid deficiency may bear an etiological relation to myotonia. Myasthenia is frequently associated with hyperthyroidism, hypoadrenalism, and probably with hyperactivity of the thymus. Hyperthyroidism and pathological findings suggesting both hypo- and hyperactivity of the thymus have been noted in amyotonia. Myopathy has been found in association with many disorders of internal secretion, perhaps most frequently with hypopituitarism. Thyroid and thymic hyperfunction are frequently observed in hypopituitarism. Whether the glandular disorders bear an etiological relation to myopathy or are secondary to it, or whether both are due to a common cause is uncertain; but the combination is too frequent to be regarded as a mere coincidence. SACHS.

Osteogenesis Imperfecta—H. C. CAMERON, Proceed. Royal Soc. Med. (London), April, 1916.

In osteogenesis imperfecta, properly so called, we have to deal with a condition characterized not only by deficient ossification and fragility of the long bones, but also by a typical and peculiar formation of the skull. These skulls are frequently confused with a hydrocephalic skull. In osteogenesis inperfecta, the skull is not enlarged, the fontanellas, although large, do not bulge, and the persistent and wide frontal suture running almost to the root of the nose is a constant feature not found in hydrocephalus of mild degree. In later childhood in cases in which life was prolonged, the most striking characteristic is a marked bulging in the temporal region sufficient to turn over the upper part of the ear. In cases of osteogenesis imperfecta, the shortening of the limbs is more marked in the proximal than in the distal portions. The author looks upon the tortuous sinuous outline of the shafts of the long bones as characteristic of this disease. SACHS.

INFECTIOUS DISEASES

The Diazo- and Urochromogen Reactions as Prognostic Aids in Pulmonary Tuberculosis—H. J. CORPER and F. F. CALLAHAN, Jour. Lab. and Clin. Med., July, 1916.

Cases dying from pulmonary tuberculosis give a positive diazo and urochromogen test at some time during the last 6 months of their illness. Whenever both tests are negative during this period death in the experience of authors was due to some intervening condition such as pulmonary hemorrhage, tuberculous meningitis, etc. Cases of chronic fibroid tuberculosis generally give both reactions negative except when same is explicable by some acute exacerbation, acute intercurrent infection or acute pleural effusion. In active cases of pulmonary tuberculosis when both reactions are positive and remain so for most of the succeeding examinations it is of grave prognostic import. When both reactions are negative in acute cases no stress can be laid upon the findings. There seems to be no regularity between the presence of either reaction, its disappearance to be displaced by the other or by both. The presence of a urochromogen reaction in cases showing no clinical symptoms is of no prognostic value. No diazo reactions were obtained by authors in clinically inactive cases, whereas a number of urochromogen reactions were obtained in such cases. The presence of a diazo or of both reactions is a *danger signal* of grave import. Careful clinical observations will, from a prognostic standpoint, give more information than the diazo and urochromogen tests in pulmonary tuberculosis. Wherever possible both tests should be performed, but of the two the diazo should be given the preference.

WESTERN.

Interpretation of Röntgenograms of the Chest in Tuberculosis—J. J. SINGER, Jour. Missouri State Med. Ass., Aug., 1916.

Author arrives at the following conclusions: A röntgenogram represents one of the most accurate aids in diagnosing lung conditions. When an area of lung tissue, normally distended with air, is not distended with air on inspiration, some pathologic condition is present—probabily tuberculosis. When the lung is distended with air, both in inspiration and expiration, we have an area of emphysema. The denser the interlobular markings the more infiltration. Cavities, whether filled with pus or broken down tissues, can readily be determined by the surrounding definite shadow and absence of the interlobular markings within. In earliest demonstrable tuberculous condition we see delicate interlobular lines approximated and apparently are so by delicate adhesions—a provision of Nature limiting the affected area to produce rest and cure—the muscle spasm over this area is analogous to abdominal inflammation with its attendant muscle spasm. When tuberculous patients are forced to breathe deeply, by exercise, high altitudes or a rapid pulse from any other condition—air is forced into these apparently closed lobules, tearing up delicate adhesions—from this separation of the alveoli, fever results with possible hemorrhage and increase of severity of the toxemia. All plates of adults show some pathologic process which coincides well with the fact that nearly all adults react to tuberculin. WESTERN.

Tuberculosis of Breast—L. DURANTE and W. C. MCCARTY, Annals of Surgery, June, 1916.

Authors observed 10 cases of tuberculosis of the breast, one of which was a male. The nipple was retracted in two cases and the skin was ulcerated in only one case. While all the cases except the man complained of some pain, in only one case was it severe. In 3 of the 10 cases, no primary focus could be clinically demonstrated, in 3 others, tuberculosis of the lungs coexisted, and in 3 others tuberculosis of the axillary lymphatic glands was present. In one case, a tuberculous pleurocostal lesion coexisted. SACHS.

Latent Syphilis and Diabetes—A. S. WARTHIN and U. F. WILSON, Am. Jour. Med. Sci., Aug., 1916.

Combined interlobular or intracinar pancreatitis with loss of the islands of Langerhans is very frequently associated with old latent syphilis. In the great majority of cases the pancreatitis is localized and patchy in character, and more rarely severe and diffuse. Latent syphilis, it seems, is the chief factor in the production of the form of pancreatitis most frequently associated with diabetes, however diabetes is not always coincident with severe degrees of this type of pancreatitis. SACHS.

Hereditary Syphilis and Spurs on Os Calcis—P. P. SWETT and H. F. STOLL, Surg. Gynecol. and Obstet., June, 1916.

Authors are of the opinion that hereditary syphilis is responsible for many cases of exostoses on the os calcis. The study of the family history is more suggestive of syphilis than examination of the patient himself. The authors studied 9 cases, 3 of which were operated on with return of the symptoms shortly after the operation. The röntgen examination aided in the diagnosis of 7 of these cases and the Wassermann test was negative in all. SACHS.

Diphtheria in the First Year of Life—J. D. ROLLESTON, Am. Jour. Dis. Child., July, 1916.

Diphtheria in the first year of life is comparatively rare. Only 20 patients at this age occurred among a total of 2,600 diphtheria patients of all ages. Congenital syphilis is an important predisposing cause. Sixty-five per cent. of the cases showed some nasal involvement with or without other diphtheritic lesions elsewhere, as compared with 25.6 per cent. in the total number of cases. Thirty-five per cent. were purely nasal as compared with 1.5 per cent. of the total cases. The mortality was high, 45 per cent. as compared with 7.3 per cent. in the total. Only 2 cases resulted in paralysis. Bronchopneumonia complicated 6 cases. A history of infection was obtained in only 3 cases. SACHS.

Pneumococcal Throat—W. CAMPBELL, Jour. Royal Army Med. Corps., June, 1916.

While ordinary septic throats and diphtheria were the most common throat conditions found in examinations made by this laboratory, mention should be made of the facts that, with the sudden event of a short period of cold weather, numerous cases of tonsillitis and pharyngitis occurred. In these cases, the temperature continued high for several days and while no suppuration occurred, the effect of this variety of sore throat upon the patient's general condition was more profound than might have been expected from the appearance of the local lesion. Many such cases were produced by a pneumococcal infection. The pneumococcal throat is a specific entity, and the likelihood of its occurrence should be borne in mind. To assist in its bacteriological investigation, it would be well to employ blood agar for the primary cultures in addition to the routine blood serum or egg medium. SACHS.

Meningitis in the Newborn and in Infants under Three Months of Age—H. KOPLIK, Arch. Pediat., July, 1916.

The prognosis of meningitis in the newborn is very grave. The streptococcus cases generally die. Cases due to other pathogenic microorganisms also die, as a rule. Though the prognosis is so fatal, author is of the opinion that the meningococcus cases, if discovered early, may be brought to a quiescent period by early and prompt treatment. The only danger is that the subsequent hydrocephalus may not limit itself, but may increase to such an extent as to cause idiocy. Lumbar puncture is most apt to meet insurmountable obstacles in the newborn, where the cerebral canals leading into the subdural space are exceedingly narrow in caliber, and at the third or fourth puncture the fluid is apt not to flow from the subarachnoid space. SACHS.

Etiology of Chorea—J. L. MORSE and C. FLOYD, Am. Jour. Dis. Child., July, 1916.

The investigations of authors show that syphilis plays no direct part in the etiology of chorea. Their results suggest that a microorganism or a group of microorganisms may be the cause of chorea.

They seem to show that if chorea is caused by a microorganism, the source of infection is ordinarily in the tonsils or teeth. They tend to confirm the belief that there is an intimate relation between chorea, rheumatism and endocarditis. SACHS.

Röntgen-Ray Diagnosis of Gas and Gas Infections—G. G. DAVIS, Surg. Gynecol. and Obstet., June, 1916.

In all cases of wounds with deeply buried bullets or shell fragments in which the röntgen-ray shows a "halo like" shadow about the missile, author found clinically at the operating table or in the laboratory, evidence of an infection caused by gas or pus forming organisms. SACHS.

Infectious Diarrhea in Infants—C. TEN BROECK and F. G. NORBURY, Bost. Med. and Surg. Jour., June 1, 1916.

The dissentery bacillus was recovered from the stools of 51 out of 75 cases of infectious diarrhea in infants. In 5 other cases while the bacillus could not be isolated in the stools, it was found in the cultures made at necropsy. SACHS.

Paralysis during Pasteur Antirabic Treatment—F. S. FIELDER, Jour. A. M. A., June 3, 1916.

(1) Paralysis complicating the Pasteur antirabic treatment is very rare, but doubtless occurs in a mild form more often than is known. It affects adults chiefly, young children almost never. (2) The prognosis is good in cases of mild or moderate degree, but the mortality among the severe cases is high. (3) The administration of antirabic treatment is therefore not entirely devoid of risk. (4) The possibility of the occurrence of paralysis in any particular case, however, is so slight that it should not deter those who evidently need antirabic treatment. A person bitten by a rabid animal is much more likely to develop rabies, which is certainly fatal, than paralysis, which usually ends in recovery. (5) The slight risk of paralysis should always be explained to those who are inclined to insist on antirabic treatment in the absence of clinical or laboratory evidence that the biting animal was rabid. (6) In most instances this paralysis is caused by the antirabic treatment itself (fixed virus infection, or toxin, or both). It is not often due to street virus infection modified by the treatment. Its incubation is shorter than that of most cases of street virus rabies. (7) Variations in the strength of the fixed virus used are probably a factor in the etiology. (8) Personal idiosyncrasy is an important predisposing condition. SACHS.

RESPIRATORY AND CIRCULATORY ORGANS

Aneurism of the Thoracic Aorta: Its Incidence, Diagnosis and Prognosis—I. I. LEMANN, Am. Jour. Med. Sci., Aug., 1916.

Author offers the following conclusions: Aneurisms of the thoracic aorta are not uncommon. Many are "latent" without direct symptoms or signs. They are a frequent cause of sudden death. The first essential to their diagnosis is alertness and an attitude of suspicion in all cases of thoracic pain and of dyspnea. The classical picture with typical signs and symptoms is characteristic only of far-advanced cases. Of all methods of physical examination percussion (of the manubrial region, the first and second intercostal spaces and of the vertebral column) has seemed to author to yield information as to the existence of a thoracid aneurism at the earliest date. The use of the fluoroscope and the röntgen rays routinely in all cases of persistent chest pain and of dyspnea without apparent cause will reveal many latent aneurisms. The early recognition of aneurisms will probably prove that the prognosis as to duration is much less unfavorable than has been hitherto thought. SACHS.

ALIMENTARY TRACT

Value of Röntgen-Ray Examinations in the Diagnosis of Cancer of the Stomach——F. H. BAETJER and J. FRIEDENWALD, Johns Hopkins Hosp. Bull., Aug., 1916.

The following summary is offered: (1) The röntgen-ray furnishes most valuable assistance as an aid in the diagnosis of gastric carcinoma; and this is especially true when the lesion is situated in the so-called silent area. It should not be relied upon alone in the diagnosis of early cases without a strict consideration of the clinical aspect of the disease, but, when taken in conjunction with the other signs, it is of the greatest diagnostic value. (2) Indurated gastric ulcers give röntgenograms similar to those of carcinoma; but inasmuch as ulcers are frequently precursors of cancer, they may be grouped for practical purposes in the same class. It is often possible to determine their true nature only by microscopic examination of the excised area. (3) In the diagnosis of carcinoma of the stomach two conditions must be taken into consideration; first, the peristaltic waves of the stomach; and, second, the morphological defect in the stomach. (4) In carcinoma of the cardiac area of the stomach there is usually a filling defect present, the activity of the stomach itself being normal, and, unless the lesion be very extensive, there is no interruption in the peristaltic waves. In lesions of the body of the stomach, the peristaltic waves are interrupted in their course at the seat of the lesion, because the lesion itself is hard and indurated, and does not admit of further peristaltic movements. There is also present a persistent filling defect. In pyloric carcinoma there are usually signs of early obstruction in addition to a filling defect of small or large size, according to the extent of the growth. (5) In carcinoma, unless there be obstruction, there is always hypermotility with rapid evacuation of contents. In ulcer there is hypermotility with pylorospasm, and more or less retention of contents. In carcinoma the filling defect is generally surrounded by an invasive area, interfering with the motility, and producing a large dead area, whereas in ulcer the filling defect is much smaller, and is not so apt to interfere with the immediate peristaltic waves. (6) The röntgen-ray furnishes us with important evidence regarding the operability or non-operability of the growth, together with the degree of obstruction and the amount of involvement. (7) Negative findings are at times quite as important as positive ones, inasmuch as absence of the various X-ray signs of cancer affords presumptive evidence of the absence of this affection.

WESTERN.

Examination of Normal Gastric Secretion by the Fractional Method—E. S. TALBOT, Jour. A. M. A., June 10, 1916.

Author believes that the fractional method of gastric analysis should replace the old tube method. The test meal must be introduced into the empty stomach. There is no one form of secretory curve common to all normal stomachs, although the vast majority of normal curves have certain characteristics in common. The emptying time of the normal stomach is fairly uniform. SACHS.

Delayed Gastric Digestion—M. E. REHFUSS, N. Y. Med. Jour., June 10, 1916.

There is a pathological deviation from the normal gastric secretory output in which there is either a delay in the appearance of the secretion, in reality delayed gastric secretion, or a delay in the evolution of the normal acid content, delayed gastric acidity resulting in delayed gastric digestion. In a large series of studies on normal subjects, this phenomenon was never encountered, indicating therefore that its appearance is unquestionably pathological.. It is not characteristic of any one type of gastric disturbance, being found in atony, gastritis, ulcer, carcinoma, and many of the functional disturbances, but its manner of evolution and the concomitant findings throw light on the disturbance. The condition, in the absence of organic disturbance, is fairly frequent and can be adequately demonstrated by the fractional method. It can be experimentally produced by the administration of hypertonic solutions, such as those of dextrose and sugar. It may or may not be associated with motility disturbances. In many of the extrinsic affections producing gastric disturbances, such as appendicitis, cholecystitis, intestinal adhesions, phenomena of delayed digestion with the ultimate evolution of high acid values and hypersecretion are found, although the initial period of gastric digestion indicates no such tendency. The explanation of delayed digestion must be found in either a disturbance of the psychic or chemical factors dominating gastric digestion, although it is probable that mucosal changes (gastritis), the elimination of toxic substances (toxemias), a disturbance in autonomic balance (nervous), a disturbance in certain internal secretions, as well as an actual disturbance in chemical equilibrium, is capable of inducing gastric secretory disturbances. Many of these cases are rebellious to treatment, others can be relieved by treatment of the underlying cause. SACHS.

Diarrhea and Vomiting of Children—E. MELLANBY, Quart. Jour. Med. (London), April, 1916.

A child suffering from diarrhea and vomiting owing to the loss of fluid, loss of bile salts, with an empty intestine and in a starving condition is in an ideal state for allowing toxic substances normally present in the alimentary canal and mucous membrane to be rapidly absorbed and have their full toxic action. The association of this disease with high atmospheric temperature in an epidemic form is to be explained largely by the additional loss of fluid due to evaporation of water whereby the child keeps down its body temperature. SACHS.

Permeability of Gastroenteric Tract of Infants to Undigested Protein—O. M. SCHLOSS and T. W. WORTHEN, Am. Jour. Dis. Child., May, 1916.

As shown by precipitin and anaphylactic tests applied to the urine, the intestinal tract of normal infants is usually impermeable to undigested foreign protein. In nutritional or gastroenteric disorders, foreign protein may be absorbed in an undigested or partially digested state and appear in the urine. The precipitin reaction, applied to the urine for the detection of egg protein, is apparently more delicate than the anaphylactic test. These results demonstrate the possibility that certain nutritional disorders in artificially fed infants may be due to the biological character of the food, although they obviously give no direct evidence to support such a view.

SACHS.

Fainting Attacks in Children—R. HUTCHINSON, Brit. Jour. Child. Dis., June, 1916.

Girls are affected with fainting attacks more frequently than boys. They occur most commonly after the school age. The child is observed to "go white," he may fall down but does not lose consciousness entirely, although in some cases he is dazed. The attacks most frequently occur in the morning before breakfast or while getting ready for school. Many of the children are nervous and dyspeptic, but in a considerable number the general health is quite good and the appearance of the child is flourishing. Author has not been able to discover any cause for the susceptibility to these attacks, but overstrain at school certainly seems to play a part in some cases. There is no reason to suppose that the heart is primarily at fault. SACHS.

Gunshot Wounds of the Solid Abdominal Viscera—C. WALLACE, Brit. Jour. Surg., April, 1916.

The symptoms of gunshot wounds of the liver are almost entirely those of hemorrhage. Many such injuries get well without operation. Many gunshot wounds of the kidneys are insignificant in themselves. The symptoms of wounds of this organ are those of hematoma and hemorrhage. Hemorrhage is more often intraperitoneal than in contusion injuries which form the greater part of the cases seen in ordinary hospital work. Next to the liver, the spleen is among the solid abdominal organs, the one most often injured by itself. There are no special symptoms that can be said to be associated with this injury. Few cases of injuries to the pancreas come under the notice of the surgeon. Two such cases proved rapidly fatal. Another case in which the bullet has passed through the organ was sent to the base hospital in good condition. SACHS.

Urticaria Tuberosa of Willan—J. A. NIXON, Quart. Jour. Med. (London), April, 1916.

Urticaria tuberosa is a rare variety of urticaria. Females are more prone to be affected. A characteristic attack is marked by the sudden rapid development of subcutaneous or deep-seated swellings, varying in size from nodules as large as a pea to tumors as big as oranges. They are distributed about the extremities. In addition to an aching pain and stiffness, tingling and burning occur, but rarely itching. The nodules usually occur at night, and disappear before morning. No complications or sequelæ have been observed. Gastrointestinal disturbances not infrequently precede or usher in an attack. Rapid recovery from an attack is the rule. Recurrence is probable and the liability to recurrence extends over many years. The disease must be distinguished from acute rheumatism. Pyrexia is either absent or very slight. SACHS.

NERVOUS SYSTEM

The Spinal-Fluid Syndromes of Nonne and Froin and their Diagnostic Significance—F. M. HANES, Am. Jour. Med. Sci., July, 1916.

Compression of the spinal cord and its meninges from whatsoever cause leads to the formation of a cul-de-sac, more or less complete, distal to the site of compression. This leads to characteristic changes in the spinal fluid. The earliest characteristic change has been described by Nonne as an increase of proteid (phase I positive) without cell increase (pleocytosis). As the condition of cord compression persists, the fluid gradually becomes yellow in color (xanthochromia), the proteïd content increases enormously, and the fluid, when removed, coagulates spontaneously (Froin's syndrome). Pleocytosis may or may not be present, depending upon whether or not the meninges are inflamed by the pathological process causing the compression. Xanthochromia of the spinal fluid must be distinguished from staining of the fluid by hemoglobin derivatives (erythrochromia. The spinal fluid syndrome of Nonne-Froin is very helpful and reliable in the diagnosis of spinal cord lesions. When present it always indicates a compression lesion of the cord.

SACHS.

URINARY ORGANS—MALE GENITALIA

The Origin of Hypernephroma of the Kidney—A. FRASER, Surg. Gynecol. and Obstet., May, 1916.

After a detailed study of 34 cases of this rather rare condition, author concludes that in 32 of his cases the tumors were derived from renal adenomata, and hence the growth would be more properly called nephromata, the term hypernephromata being reserved for malignant tumors arising from cortical adrenal cells. He further concludes (a) that the primary structures of tumors of adrenal origin are essentially different from the primary structures of tumors of renal origin; (b) that the primary structures of adrenal tumors never imitate the primary structures of renal tumors; (c) but the primary structures of some renal tumors (adenomata) can, at an early stage, through proliferative changes, imitate the primary structures of tumors of adrenal origin; (d) both tumors of adrenal and renal origin can undergo secondary degenerative and malignant changes, which make their histological features very similar, these changes being practically always present in the renal tumors. Finally he believes that the diagnosis should not be based on the appearance presented by those parts of the tumor that have undergone secondary degenerative and malignant changes.

HELLMAN.

Aspermatism—V. D. LESPINASSE, Urolog. and Cutan. Rev., July, 1916.

Author reports the following interesting case of aspermatism. (Aspermatism as defined by Roubaud in 1855 is characterized by the failure of ejaculation with a normal erection as against priapism and with neither perversion or exaltation of the normal faculties as against erothromania.) Author's patient came to him complaining that the act of intercourse was never satisfactorily finished. He is married and has never attempted intercourse with any one but his wife and has had this trouble throughout his entire married life. He has never been sick, is in perfect general health, an automobile salesman by occupation, has never had any venereal disease. Examination shows a young man 25 years old of normal nutrition and of a healthy color. The external genitals are normal as to size, development and general appearance. Testicles are of normal size, normal tension and both hang in the scrotum as they should. Penis is of normal size, urine is clear and free from albumen and sugar. Rectal examination shows the prostate, vesicles and bulb of the urethra normal. Cystoscopic and urethroscopic examinations reveal nothing pathological. He has normal sexual desire, normal erections, normal penetration and normal intercourse in every way up to the point of ejaculation. No matter how long or how vigorously the act is carried out or what psychic aids are brought into play, ejaculation never takes place and the act is concluded when both he and his wife are physically exhausted or simply stop. The patient has dreams which are accompanied by emissions and the fluid discharged contains prostatic fluid, spermatozoa and all the elements that a normal man would pass under similar circumstances.

WESTERN.

Prognosis of Pyelocystitis in Infancy—C. G. GRULEE, Urolog. and Cutan. Rev., May, 1916.

Pyelocystitis is usually looked upon as a condition which, though running a high temperature, gives an almost absolutely good prognosis. Author's experience with these cases has been quite to the contrary. While it is true that a large proportion do recover without serious consequences, it is also true that a very great number have repeated attacks of the infection and that not a small proportion of these infants succumb to the disease, either in the acute stages or later. Death in these cases may occur during a very acute onset, or the condition may become sub-chronic, existing for several weeks, the infection spreading and the child gradually succumbing to a sepsis. Or the condition may so deplete the infant's organism that a marasmus of parenteral cause is produced and the child succumbs to some intercurrent infection. Much more likely than a fatal termination is the tendency to repeated attacks, and no man who has seen many of these cases but is impressed with the fact that no matter how thorough the treatment may be according to the lines laid down at present, repeated attacks are very common. The attacks, as a rule, tend to become less severe but one can never feel satisfied that a case of pyelocystitis is effectively cured and that there is not present at least a tendency, probably a focus of infection, which will result in a second, a third, or more attacks. It would seem to author not at all unlikely that some cases of pyelocystitis of pregnancy might be due to this condition, and that the infection existed with repetitions, the nature of which had not been recognized during the intervening years. The danger of a chronic nephritis resulting from pyelocystitis in infancy is relatively slight. Of 73 cases of chronic nephritis in infancy and childhood which Heubner was able to collect from his records (of 17,000 cases), only six were due to pyelocystitis, in his opinion. In closing author wishes to lay emphasis on the fact that pyelocystitis is a common condition in infancy, that frequently its only symptom is fever, the cause of which is determined only when the urine is examined; that the condition is one whose seriousness has been greatly underestimated, one which not infrequently leads to death, and which has a marked tendency to repetition in a large proportion of cases.

WESTERN.

Gonorrheal Complement Fixation Test—V. D. LESPINASSE, Urolog. and Cutan. Rev., April, 1916.

The gonorrhea complement fixation test is of great value in the diagnosis of some phases of gonorrhea. The test errs on the negative side more than on the positive. A positive result means gonorrhea; a negative result means nothing. Like all laboratory tests, the test should be given its full value, but it should not be overestimated, or under any circumstances be considered absolute.

WESTERN.

FEMALE ORGANS OF GENERATION—PREGNANCY—PARTURITION—INFANTS

A Speculum-Camera—H. R. SPENCER and E. A. BARTON, Proceed. Royal Soc. Med., March, 1916.

In the authors' instrument, a camera and speculum are combined and the focus is fixed. The instrument measures 8¾ inches in length. The speculum is passed into the vagina while the patient lies on the left side and the projecting end is supported by sand bags. Photographs of diseased cervices are shown in this article.

SACHS.

A Study of Liver Function in Normal Pregnancy—J. C. LITZENBERG, Am. Jour. Obstet., Feb., 1916.

To test the liver function in pregnancy author selected the urobilinogen and urobilin test because he considered it as nearly specific as any, and because of its simplicity of application. The test is made by adding 5 drops of Ehrlich's solution to 5 c.c. of urine and leaving it in a dark place for at least fifteen minutes, when, if positive, a distinct pink or deep red color will appear. To make sure that the red color is due to urobilinogen, shake with chloroform; if urobilinogen is present the chloroform will become red. To test for urobilin 10 c.c. of Schlesinger's solution is added to 10 c.c. of urine filtered. If urobilin is present a green fluorescence will be seen which is best brought out by throwing condensed light through the fluid by a lens or flask of water. The urine of 271 women was examined. Seventy-one were normal non-pregnant women, and the tests were uniformly negative. Of the 200 seemingly normal pregnant women 62 (31 per cent.) gave the reaction for urobilinogen or urobilin. Now, if these substances in the urine show, as is believed, either increased blood destruction or inefficiency of the liver, we must come to the conclusion that there must frequently be a disturbance of liver function during pregnancy. HELLMAN.

Pyelitis of Pregnancy with Especial Reference to its Etiology—W. C. DANFORTH, Surg. Gynecol. and Obstet., May, 1916.

To determine the etiology of this condition author experimented with urine from pregnant women. The specimens were all obtained under the most aseptic conditions. In the first series of 50 women the urine showed a pure culture of staphylococcus 32 times, colon bacillus in pure culture twice, and 3 cases gave cultures of both these organisms. The second series of 14 cases showed staphylococcus 8 times. Four were entirely negative. In 2 cases of pyelitis the ureteral urine showed pure cultures of colon bacillus. Author believes that in many, if not all, pyelitis cases, the infection which is most often the colon bacillus, is a blood-borne infection and not an extension from the bladder. There is a most excellent bibliography appended to the article. HELLMAN.

Pyelitis in Pregnancy—J. C. WEBSTER, Urolog. and Cutan. Rev., May, 1916.

The symptoms and signs of the disease are the same as in non-pregnant women. In some cases the patient may complain only of malaise and slight fever, without any pain. The fever may be high and may be accompanied with chills. There is often aching in the loins. Frequently attention is first called to a pain in the affected side, accompanied by fever, nausea or vomiting; it may be anywhere along the urinary tract. In some cases there is frequency of urination. Rarely is there actual bladder distress, except where this viscus is involved. When the disease occurs on the right side, the pain may simulate that found in appendicitis or even in gall-bladder disease. Serious mistakes have been made in diagnosing these cases as appendicitis, cholecystitis and adnexal disease and in opening the abdomen for the relief of the condition. During one month, a few years ago, author was asked to operate several times for appendicitis in pregnancy after the sixth month where acute pyeloureteritis alone existed. In each instance vaginal examination had not been made and urinalysis had been neglected. In all cases these two procedures are of the greatest importance. In many there is very definite tenderness along the lower portion of the affected ureter which may be so thickened as to be readily palpable. The most marked outlining of the ureter occurs when a stone is impacted and surrounded by fibrin and in tuberculous infiltration. In the urine there is albumin and pus, epithelium from the pelvis of the kidney or ureter. In some stages red blood corpuscles are found and when the true kidney substance is involved, there may be considerable serum albumin and casts. The quantity of urine tends to be decreased and its specific gravity increased. Micro-organisms are usually abundant except possibly in the very early stage of the disease, and of course variations are found from time to time in long protracted chronic cases. Cystoscopy and ureteral catheterization are rarely ever necessary to establish the existence of the disease, but may be helpful if it be thought important to determine the extent of the urinary tract involved. WESTERN.

A Statistical Study of 635 Labors with the Occiput Posterior—E. D. PLASS, Johns Hopkins Hospital Bull., June, 1916.

After a most detailed, statistical and arithmetical study of 635 cases of occiput posterior that occurred in 7,500 consecutive deliveries author concludes: (1) The frequency of occiput obliquely and directly posterior positions was 11.57 per cent. in the series, although the actual percentage of cases originally in this position was probably considerably higher. (2) Rotation of the occiput into the hollow of the sacrum occurred in 14 per cent. of the cases. (3) Three factors which tend to favor rotation into the hollow of the sacrum are poor flexion, small size of the head, and funnel pelvis. Contraction of the pelvic inlet favors anterior rotation. (4) The etiology of internal rotation is not satisfactory, but Hodge's theory of the ischial spines and inclined planes is the most acceptable. (5) Premature rupture of the membranes was not especially frequent when the occiput was posterior. (6) Spontaneous delivery was the usual outcome. The total operative incidence was 22.9 per cent., but in only 11.02 per cent. was the position possibly accountable for the need for intervention. (7) There was no increased infant mortality because of the posterior position. (8) Labor was not prolonged, independently of whether the occiput rotated to the symphisis or into the hollow of the sacrum. (9) A contracted pelvis did not increase the incidence of the posterior positions. (10) There was no additional maternal morbidity or mortality.

HELLMAN.

Effects of Nutrition of Mother on Child—G. F. D. SMITH, Lancet, July 8, 1916.

Statistics recited by the author seem to suggest the following conclusions: A state of bad nutrition of the mother at the time of labor due to insufficient food (1) greatly increases the percentage of dead births, (2) greatly increases the percentage of premature births, (3) slightly decreases the average weight of the full-time baby at birth, (4) definitely increases the post-natal infantile mortality, (5) has little if any effect during the first eight or ten days on the progress of the babies who live during that time and possibly increases the death rate of babies during the first three or four days of life. A state of good nutrition of the mother at the time of labor, on the other hand, (1) considerably increases the average weight of the full-time baby at birth and (2) increases the percentage of mothers who are able to suckle during the first eight or ten days of the puerperium, quite apart from any effect from the use of an ample diet during this time. SACHS.

Recent Publications

THE DUCTLESS GLANDULAR DISEASES. By WILHELM FALTA, Vienna. Translated and edited by Milton K. Meyers, M.D., Neurologist to the Lebanon Hospital, and to the Dispensaries of the Jewish and St. Agnes Hospitals, Philadelphia, etc., etc. With a Foreword by Archibald E. Garrod, M.D. (Oxon.), F.R.C.P. (London), F.R.S., Physician to St. Bartholomew's Hospital, London. Second Edition. With 101 Illustrations in the Text. Philadelphia: P. Blakiston's Son & Co.

This work of Falta is a classic. This means that we possess nothing like it which treats with the clinical side of affections of the ductless glands in such masterly manner. There are more voluminuos works on the subject, but there exists no treatise which is freer from hypothesis and speculation than Falta's.

Whoever wishes to be abreast of the times should purchase this volume which the reviewer indorses unconditionally. T. F. R.

THE TREATMENT OF INFANTILE PARALYSIS. By ROBERT W. LOVETT, M.D., John B. and Buckminster Brown Professor of Orthopedic Surgery, Harvard Medical School; Surgeon to the Children's Hospital, Boston; Surgeon-in-Chief to the Massachusetts Hospital School, Canton; Consulting Orthopedic Surgeon to the Boston Dispensary; Member of the American Orthopedic Association; Corresponding Member of the Royal Society of Physicians, Budapest; Korrespondierendes Mitglied der deutschen Gesellschaft für Orthopädische Chirurgie, Socio della Societá Italiana di Ortopedia. With 113 Illustrations. Philadelphia: P. Blakiston's Son & Co.

The major part of Dr. Lovett's book is devoted to the treatment of the chronic phase of poliomyelitis. This is quite natural as we practically know nothing about the treatment of the acute stage.

So far as the chronic phase of the infection is concerned the orthopedist is still master of the situation. The author has given us a clear outline of all the mechanical methods of treatment and his book can be highly recommended to those who come face to face with any of the chronic phases of the affection. T. F. R.

THE EXPECTANT MOTHER. By SAMUEL WYLLIS BANDLER, M.D., Professor of Gynecology in the New York Post-Graduate Medical School and Hospital. Illustrated. Philadelphia and London: W. B. Saunders Company, 1916.

This is a thoroughly scientific book of 213 pages which is written in language that is understandable by every intelligent person. Although the subject matter is not arranged in alphabetical order I would call the book "The Expectant Mother's Encyclopedia," for it sheds light upon practically everything a young woman needs to know about motherhood.

Books for the laity, like this one, must be written by masters in their respective fields. Dr. Bandler is such a master. H. W.

THE

ARCHIVES OF DIAGNOSIS

Editor, Heinrich Stern, M.D., LL.D.
250 West 73d Street, New York, N. Y.
To whom all Communications of an *Editorial* Nature should be addressed

Publishers: Rebman Company
141, 143 and 145 West 36th St., New York, N. Y.

PRICE OF SUBSCRIPTION PER YEAR
(payable in advance)
To Subscribers in the United States, Canada and Mexico........... $2.00
To Subscribers in Other Countries (Postage prepaid)............... $2.50
Price of Single Copies, 75 Cents
The volume consisting of four issues comprises at least 400 pages

NOTICE TO CONTRIBUTORS

1. *The purport of the Archives is to publish papers which embody the results of original work and observation pertaining to Diagnosis and Prognosis.*
2. *Papers treating on Early Diagnosis, Functional Diagnosis, Therapeutic Diagnosis and Differential Diagnosis are especially solicited.*
3. *Contributions must not be of unreasonable length.*
4. *No contribution that has already appeared in print will be accepted for publication.*
5. *Galley Proof and Page Proof will in all instances be sent to the author for correction.*
6. *Contributors will receive 200 copies of their papers in pamphlet form free of charge. Additional copies will be furnished at cost.*

THE

ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX OCTOBER, 1916 No. 4

FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York

PUBLISHED BY
REBMAN COMPANY
141, 143 AND 145 WEST 36TH STREET
NEW YORK, N.Y.

COPYRIGHT 1916 BY REBMAN COMPANY. ALL RIGHTS RESERVED.

Entered as Second-Class Matter, February 8, 1908, at the Post-Office at New York, N. Y.
Under the Act of Congress of March 3, 1879.

SUBSCRIPTION TWO DOLLARS A YEAR. SINGLE COPIES 75 CENTS. FOREIGN $2.50.

THE
ARCHIVES OF DIAGNOSIS
A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. IX OCTOBER, 1916 No. 4

Special Articles

CLINICAL DIAGNOSIS OF LUETIC AORTITIS

By I. J. LEVY
Assistant Pathologist, City Hospital; Adjunct Physician, Sydenham Hospital
New York

It is now acknowledged by clinicians that syphilis is the underlying cause in organic changes in the aorta with secondary involvement of the heart in a greater number of instances than was generally supposed. In a disease in which such destructive changes can occur in an organ in a period of about twenty years, in many cases in less time than that, it is important that we turn our attention to its early diagnosis; that is, recognition of the disease before the anatomical destruction is beyond repair. It is convenient, therefore, to divide the course of the disease into clinical periods, not arbitrarily arranged, but based upon different stages in its pathological development.

Further, luetic aortitis is a productive inflammation passing on to a stage of fibrosis, with an end stage of sclerosis and degeneration. Depending upon the portion of the aorta attacked and the organs secondarily involved, the clinical picture varies and gives rise to different clinical types.

The most constant symptom of syphilitic aortitis is a positive Wassermann reaction. Since in recent years the literature points out numerous discrepancies in the results of the serologists, undoubtedly due to different antigens and various methods employed, it becomes necessary not only to report the serological diagnosis as "positive" or "negative," but to state further the method of procedure. That each method has its advantages is not to be denied. The clinician must now learn to recognize which serological procedure is to be preferred in diagnosing the skin, visceral and nervous manifestation of syphilis. Recently Larkin and myself have shown, when employing a technic differing only slightly from the original Wassermann, that, in individuals with positive reactions suffering with syphilis for a period of about fifteen years, in ninety per cent. of the cases we could demonstrate histological luetic changes in the aorta, and, further that luetic aortitis gave ninety-four per cent. of positive fixations. This has now been adopted as the routine method in performing the Wassermann reactions, as distinguished from the other methods employed at the Strecker Memorial Laboratory at the City Hospital. When using other methods, employing sensitized cholesterin antigens, or cold fixations, etc., we can undoubtedly obtain a higher percentage of positive reactions in other types of syphilis. In a series of cases, with the sensitized antigens, however, only seventy-seven per cent. of the positive reactions showed evidence of syphilis in the aorta. Keeping these facts in mind I would suggest that, when substantiating a diagnosis of luetic aortitis a positive fixation with the "routine method" be accepted as absolutely corroborative. If negative, the sensitized antigens may be resorted to, and a positive reaction interpreted as only fair evidence in favor of a diagnosis of aortitis. If the patient has undergone treatment, the fixations with the sensitized antigens are of much more value. Often, only after short periods of antisyphilitic treatment a positive reaction with the "routine method' may be rendered negative, and in such a case a positive fixation with sensitized antigens only, should carry more weight in corroborating a diagnosis of syphilitic aortitis, or for that matter, any syphilitic lesion.

The age of the patient is an important factor in the diagnosis. Most authors agree that the patient presents unmistakable evidence of the disease at about the fortieth or fiftieth year. Stadler states that about twenty years elapse between the date of infection and death. He has seen a few cases run their course in five years, and Brooks in as short a period as two. In a series of cases which have come to autopsy the average age at death was forty years. How soon after the infection the aorta is attacked is difficult to say, but surely years may elapse before the disease presents any symptoms. It must also be borne in mind that hereditary syphilis plays an important rôle, and that the syphilitic involvement of the aorta may begin at birth.

From the foregoing—the pathology and long latent period of the disease—there is good reason to divide the course of the disease into three clinical periods.

(1) Primary stage, or latent period.
(2) Advanced stage.
(3) Final stage.

PRIMARY STAGE

The patients usually complain of no subjective symptoms, and physical examination of the chest adds nothing to our information, nor does the X-ray reveal any abnormalities in the aortic shadow. Pain in the chest receiving only passing notice of the patient may be the only complaint which brings him to his physician. A positive Wassermann reaction, however, in an individual in the early forties complaining of indefinite chest pains should be strong presumptive evidence of an early productive inflammatory process in his aorta. The fact that ninety per cent. of individuals with positive serology have evidence of luetic changes in their aorta, providing the disease has been present for a period of fifteen years, and that sixty per cent. of these die of their aortitis, makes it clear though the diagnosis may be correct in ninety cases in a hundred, that forty per cent. of these individuals may live on and die of other causes, the diagnosis only being substantiated at autopsy by histological examination of the aorta. But since there is no method of differentiating which patients are candidates for the final stage of the disease we must look upon all with suspicion. This is of the utmost importance, for if antispecific therapy is to be of any avail in luetic aortitis, it is only in this primary stage that we can hope to obtain definite and lasting results.

ADVANCED STAGE

This period of the disease, too, may present few if any symptoms. The decreased elasticity of the aorta is usually compensated for by the concentric hypertrophy of the heart, and the patient may remain in apparent health for a number of years. Angina-like pains, or a true angina, may be the only warning signal of impending trouble. The pain is caused, as Longcope has suggested, by the inflammatory process around the small nerve fibrillæ in the aorta, and not always by coronary sclerosis, as has been generally supposed. It is often the experience of recent pathologists to find normal coronary arteries in individuals who during life have suffered with severe anginal pains. Dyspnea on exertion and palpitation are among the early symptoms, due to a latent cardiac decompensation long before there is physical evidence of a decompensating heart. Stadler states that a common complaint is a general physical weakness, or the patients present themselves with various neurasthenic manifestations. It is probably late in this stage of the disease that the aortic valve becomes involved, or, at least, the involvement becomes extensive enough to give clinical evidence of an impaired valve. This is discovered accidentally during a routine physical examination, or when in quest of an explanation of a positive Wassermann reaction or angina pains. A diffuse dilatation of the aorta, as shown by the X-ray, is further corroborative evidence, but a positive Wassermann reaction is practically obligatory for a reliable diagnosis.

FINAL STAGE

It is in this period that we observe the classical clinical picture of luetic aortitis. The disease has run its pathological course, resulting in a sclerosed aorta, with diffuse dilatation or aneurism, coronary sclerosis and aortic insufficiency. The heart is only secondarily involved, depending upon the grade of coronary sclerosis and the presence or absence of disease of the aortic cusps.

The angina pains come oftener and are much more severe. The dyspnea now is associated with edema of the lower extremities. The patient may be entirely free of all symptoms and suddenly complain of paroxysmal attacks of dispnea at night (nächtliches Asthma) coming on quite suddenly during sleep. When in an acute attack the distress of the patient and the physical signs of the chest are not to be differentiated from true bronchial asthma, but the attack coming on late in life with a history of no previous trouble is enough to warrant the conclusion that it is of cardiac or rather of aortic origin. Though this is quite characteristic of luetic aortitis it is not entirely confined to this condition; it is also noted in cardiac decompensation of a benign hypertension, and in the presence of hypertension of a secondary contracted kidney. Marked edema is a late symptom and is the result of a decompensating myocardium.

Diffuse dilatation of the aorta is an important factor in the diagnosis. It can readily be ascertained by percussion of the anterior wall of the chest or by X-ray or fluoroscopic examination of the thorax. On auscultation there is a characteristic ringing quality to the second aortic sound which can be easily differentiated from a second accentuated sound so readily discernible in hypertension cases. It is to be remembered, however, that a diffuse aortic dilation is not necessarily the result of a syphilitic process, for in general arteriosclerosis the aorta may present the same picture. In hospitals when patients present themselves with well-marked evidence of cardiac decompensation, and when the aortic valve has escaped the ravages of the disease and the X-ray picture of the thorax presents only a diffuse dilatation of the aorta, there may be some difficulty in deciding the cause for the vascular changes. A positive Wassermann reaction, if the specific infection is not of recent origin, should be accepted as practically conclusive evidence. But it is not to be overlooked that an individual with a decompensating heart, resulting from a general arteriosclerosis, may coincidentally have lues, though the syphilis in no sense is a cause of his vascular changes.

AORTIC INSUFFICIENCY

The cases with aortic insufficiency are much easier to diagnose. It is now generally established that pure aortic insufficiency is the result either of acquired or inherited syphilis, with the exception of an infectious endocarditis attacking only the aortic cusps. In a series of cases at autopsy the syphilitic aortic lesions gave one-hundredth per cent. positive Wassermann reactions on the blood antemortem.

It is not necessary here to discuss the type of murmur or the characteristic enlargement of the heart associated with aortic insufficiency, but only to emphasize that the diastolic murmur is often more distinctly audible over the mitral area. The differentiation from an infectious endocarditis may present some difficulty. Occasionally the vegetations are found only on the aortic valve, and damage resulting in an insufficient cusp may render the possibility of a positive diagnosis extremely difficult. Repeated negative blood cultures and positive serology are strong presumptive evidence in favor of syphilis. Recently I saw a most unusual case come to autopsy which had been diagnosed as luetic aortitis. The patient was a girl of about thirty years, with aortic insufficiency, who died with cardiac decompensation. Several blood cultures were negative. The Wassermann reaction was positive on two separate occasions. At autopsy the heart revealed a partially destroyed aortic valve with vegations on its ventral surface. The positive Wassermann was explained, however, by early syphilitic changes in the aorta. Longcope speaks of two cases autopsied in which an endocardial infection of both valves was superimposed upon an arterial aortic insufficiency. Of course an absolute diagnosis in such a case is also very difficult.

When general arteriosclerosis attacking both valves is superimposed upon a syphilitic process, the syphilitic basis of the disease in the arterial system may be easily overlooked, but a positive Wassermann reaction should guide one against such an error.

ANEURISM

Briefly, an aneurism is a sacculated as opposed to a diffuse dilatation of the aorta. The ascending and transverse portions of the aorta are the usual sites, but it is by no means uncommon to have the thoracic and upper half of the abdominal portion involved. Of course the symptoms are directly dependent upon the part of the organ attacked. The ones most easily recognized are those in the ascending and transverse portions. In most patients dying of aortitis the aorta presents aneurismal dilatations which may give no characteristic symptoms and are discovered only by an X-ray examination. Usually, however, one or more signs referable to the aneurism are readily elicited. When involving the first portion of the aorta the diagnosis can often be made by the presence of a "tracheal tug." The tugging on the trachea is undoubtedly due to a thick band of adhesions between the trachea and aorta. Pressure upon the phrenic nerve, esophagus or lungs produces its train of symptoms, percussion reveals an intra-thoracic tumor which often is easily recognized by diffuse pulsation of the anterior chest wall or in the supra-clavicular area. Patients are often admitted to the hospital with symptoms of cardiac decompensation and an aneurism is discovered in a routine examination. They are often seen with a series of complaints common to the second stage of the disease, or with no symptoms referable to the aortitis, die suddenly from a rupture of an aneurism. It is interesting to note at autopsy how in this type the process has localized itself to a definite portion of the aorta while the coronary arteries and aortic valves have practically escaped.

In an aneurism of the lower thoracic portion, erosion of the adjacent vertebra usually causes a dull boring or marked excruciating pain. It is surprising, however, that even with the bone eroded almost to the cord how little pain the patient may suffer.

An X-ray examination usually makes a definite diagnosis, and it is of much more value than when showing a diffuse dilatation, for in shadows showing sacculations syphilis is always the underlying cause of the pathological process whereas in the diffuse type it is difficult to say whether the etiological factor is syphilis or arteriosclerosis.

COURSE AND PROGNOSIS

The latent interval and chronicity of the disease, then, is the striking feature of aortitis. The patient usually is in apparent health for years and is suddenly stricken with serious symptoms in the prime of life. When the cardia has once broken under the strain and the patient is edematous, dyspneic and bedridden, one sees little response to rest and digitalis, and death supervenes in the course of a few years. A heart, laboring with an endocardial infection may present this picture several times during life, but each time responds to treatment before the final outcome. A luetic endarteritis may leave the patient paralyzed before cardiac decompensation supervenes, and undoubtedly in a number of cases general paresis or tabes is the closing scene in a latent aortitis.

REFERENCES

1. Brooks, H.—Med. Rec., New York, xxxi, l 351, 1913
2. Larkin and Levy.—Pathological Study of Syphilitic Aortitis and Its Serology, Jour. of Exper. Med., vol. xxiii, No. 1, 1916.
3. Longscope, W. T.—Syphilitic Aortitis: Its Diagnosis and Treatment, Arch. Int. Med., vol. xi, 1913.
4. Stadler, E.—Die Klinik der Syphilitischen Aortenerkrankung, Jena, 1912.

THE CLINICAL AND LABORATORY MANIFESTATIONS OF MALIGNANCY

BY ARNOLD STURMDORF

Clinical Professor of Gynecology, New York Polyclinic Medical School and Hospital, etc.

The study of the problem of malignancy is world wide; in nearly every civilized land special institutes exist for cancer research, some of them munificently endowed by legacies from the very victims of this dread disease; an eager quest for cause and cure has dominated the keenest investigators for many years, nevertheless, with theories too numerous to mention, our attitude today, as practitioners in this morbid problem, is well defined by "Empodecles on Etna."

"The gods laugh in their sleeve
To watch man doubt and fear,
Who knows not what to believe
Since he sees nothing clear,
And dares stamp nothing false
Where he finds nothing sure."

But there is profitable sifting in this maze of facts and fancies, so let us arrange these siftings, as to balance theories with conditions, giving to each its due weight in the scales of clinical experience.

It will appear reminiscent of the symbolystic age to state that three monosyllables embody the enigma of life: the study of its every vital phenomenon and the solution of all its morbid manifestations.

What! Whence! Why! What is cancer? Whence comes cancer? Why is cancer?

He who solves this Gordian triad unveils the Isis of malignancy.

It must seem incredible to the uninitiated that the whole range of tumors, both benign and malignant, is composed of elements normal to the various tissues of the body, in other words that every variety of new growth presents only normal elements, abnormally disposed; or, to put it technically, "the epithelial cells of any given tumor are the histogenetic offsprings of the tissues from which such tumor is developed."

This conforms to an inflexible principle in biology known as "The law of prototypes," whose recognition in the histogenesis of tumors marks the establishment of the *first* fundamental fact in the cancer problem.

It enables the pathologist to predicate with certainty the various types of primary growth to which a tissue or organ may give rise and enables him to explain why a tumor containing epithelium of one character cannot spring from tissues presenting epithelium of a different character; in other words, "like epithelium can only generate like epithelium"; a connective tissue cell can no more give birth to a ciliated epithelium than one species could generate another.

The widest dissemination and the remotest metastatic deposits of a tumor present the cytologic features of the parent growth. So faithful is this reproduction that the nature of the primary tumor can often be correctly inferred from the microscopic examination of a secondary nodule; thus it is astonishing to find a metastatic deposit in the humerus with all the characters of the rectal glands, a multitude of secondary nodules in the ovary or skin with the structural features of gastric glands, or nodules resembling mammary carcinoma, in the brain, eye or spinal cord.

This law of prototypes inaugurated the modern histological classification of tumors, a classification, however, which clinicians soon relegated to the academic routine of the pathological laboratory, adopting in preference the more practical bedside distinction, which recognizes only the benign or malignant tendencies manifested by a given neoplasm.

The more closely the histology of tumors is investigated the greater the difficulty to establish a borderline between the benign and malignant.

Few tumors had a better laboratory stamp of innocence than the dermoids and testicular teratomata, and yet we know that, clinically, they may prove the most malignant growths that attack mankind.

History repeats itself in medicine as in all else, and the histological classification of the pathologist is giving way to the bedside distinction of the clinician, for we possess today conclusive evidence that *all* forms of tumor, that is, every abnormal disposition of tissue elements or epithelia, are possessed of malignant potentialities in local and constitutional manifestations, histologically benign growths frequently exhibiting all the destructive tendencies of typical cancers.

Let us adduce some familiar clinical corroboration of the foregoing.

During one period of embryonic life the supra-renal bodies or adrenals inclose nearly the whole upper half of what, at that time, is the distinctly lobulated fetal kidney, and it occasionally happens that small portions of such adrenals become included in the crevices between the growing kidney lobules; thus we find in later life larger or smaller masses of adrenal tissue beneath the adult kidney capsule.

Such tissue bears the distinctive characters that readily identify it as composed of normal adrenal structures.

In the majority of cases these adrenal inclusions remain innocuous throughout life, but in some instances they assume an active and abnormal growth, with all the manifestations of the most virulent malignancy.

Such hypernephromata exhibit in their structural features two distinct types of malignant formation, namely, sarcomatous and carcinomatous—but, since the tissues from which they originate present both connective tissue cells and epithelia, the histological explanation for this apparent disparity is not difficult.

The clinical course and characters of these tumors is most perplexing. We have already noted that they may remain small, benign and unproductive of any symptoms for many years or a lifetime.

On the other hand, they may grow slowly, reaching enormous dimensions, with only local symptoms, such as would occur with any large, simple growth; but, in some cases, the smallest nodule suddenly becomes active and then—either by direct invasion of the renal pelvis or extension into the large veins—causes a sudden, copious and at times fatal hematuria.

Once their malignant potentialities are activated, these growths produce metastases of a most dangerous and fatal character, marked by profound anemia and general toxemia.

The curious and inexplicable phenomenon is presented of a simultaneous benign and malignant dissemination, the original growth maintaining all of its innocuous characters while some of the metastases are most actively malignant.

Cases are on record where the histological diagnosis of a malignant hypernephroma, removed from one of the long bones, prompted exploration and nephrectomy for a previously unsuspected hypernephroma of the kidney.

Without multiplying examples, we have in this instance alone a single morbid entity, presenting all the histological and clinical variants that characterize tumors as a class, composed of elements normal to the bodily structures, but abnormally disposed and distributed, existing in the same individual as quiescent deposits in one situation and as actively virulent growths in another, presenting the microscopic features of an innocent nodule in one locality or of sarcoma here and of carcinoma there; the same tumor elements that prove perfectly harmless throughout a long life of one host being rapidly destructive of another.

Such riot of vital aberrations, all focussing attention upon developmental phases in tissue generation, led Cohnheim to promulgate his embryonic theory of cancer.

This brilliant generalization has dominated the medical mind since 1882. It assumes that *all* tumors, benign and malignant, originate in certain unutilized aberrant residues of embryonic cells, termed rests or tumor germs, which either remain quiescent or develop into pathological growths during any period of their existence, from fetal life to old age.

Cohnheim's theory was conceived to illuminate the etiology of cancer and remained to serve as the key to the genesis of embryomata. While it proved most fruitful in directing a line of inquiry that has afforded the most important data on the pathogenesis of dermoids and allied inclusions, it must be discarded as explanatory of cancer, first and foremost because the so-called germinal rests, upon whose existence the whole theory is founded, have not been demonstrated, secondly because it is entirely inadequate to explain the occurrence of that large group of carcinomata, which involves the flat surface epithelia known to pathologists as squamous cells.

It does not explain the occurrence of accidental implantation cancers, as exemplified by the virulent growths that occur in otherwise normal laparotomy scars after extirpation of ovarian adenomata and papillary cystomata.

If we recall that cancer in its initial manifestations is a strictly localized disease; that it spreads by continuity, infects the lymph channels and blood vessels thus becoming disseminated throughout the body, inducing a profound toxemia; that it may remain dormant for years, the primary focus sometimes disappearing, as in some forms of mammary scirrhus, while secondary deposits assume virulent activities; that it evidently undergoes an undefined period of incubation; we construct the clinical picture of a chronic infection, analogous to that of syphilis, tuberculosis, leprosy and similar entities.

This suggestive parallelism, in the clinical course of cancer and the diseases of established bacterial origin, naturally offered the incentive to an ardent search for a specific germ, but to the present time none of the alleged discoveries have produced an organism that fulfils even one of the three postulates essential to establish the bacterial nature of cancer.

1. The alleged germ was not shown to be present in all cases.
2. It was not susceptible of individual cultivation.
3. It failed to reproduce the original disease when inoculated into a healthy animal.

The refinements of tinctorial differentiation introduced by modern bacteriology, while so far unsuccessful in isolating a specific bacterium, have nevertheless disclosed two important phases in the cancer problem. In the first place, we have learned that in the disseminated foci of bacterial disease we find a transferrence of the original *specific infecting bacterium,* while in the metastases of malignant foci the metastatic deposit presents the transferred *specific epithelium* characteristic of the original tumor, in other words, the epithelial cell itself represents the infectious and infecting cancer organism.

Furthermore, certain interesting cytologic transformations have been revealed as existing within these epithelial cells, the significance of which is as yet a matter within the realm of speculation.

It has long been known to biologists that in the production of sexual cells in plants and animals the process of nuclear cleavage or segmentation differs materially and in a highly complex manner from that regularly observed during the production of ordinary tissue cells and it has been possible for Farmer, Moore and Walker to detect and trace in detail a number of definite serial changes in the epithelia of proliferating malignant tissue, which are strikingly similar to those occurring during the maturation of the cellular elements contained within the sexual reproductive glands. This resemblance extends to the minutest points of detail.

With these facts once established, the same investigators soon succeeded in demonstrating that the remarkable vesicular structures found in cancer epithelia, known as "bird's eye inclusions" or Plimmer's bodies and hitherto alleged to represent the specific cancer protozoon, occur normally during the production of sexual cell elements in all vertebrata.

This exposure of the spermatogenetic origin of "Plimmer's bodies" at once deposed the last of the germ candidates from the bacteriological field of the cancer problem and inaugurated the present dawn of what may be termed the era of epithelial doctrine.

Thus our attention has been turned again from the germ to the cell, from bacteriology to cytology.

Epithelial overgrowth, as the result of known irritants, chemic, thermic, mechanic or bacteriologic, are familiar daily clinical phenomena; as example may be cited the ordinary exuberance of granulation tissue observed in wounds of various nature; the tumor masses known as "infectious granulomata," such as occur in epulis and actinomycosis; hypertrophic scars, keloids, X-ray burns and other conditions presenting the various disturbances in the healing process, many of them exhibiting a marked proneness to ultimate malignancy.

Epithelium will thrive and multiply wherever parent cell and pabulum exist. We take advantage of this in skin grafting.

Accidental implantation of dermal epithelium or hair bulbs into the underlying connective tissue will result in so-called implantation cysts, such as are commonly found on the fingers of seamstresses from needle punctures, on the cornea, in the iris and other exposed parts.

We have already alluded to implantation cancer in laparotomy scars following extirpation of adenomatous ovaries. Such implantation cancers of the abdominal wall present small cysts, furnished with the regular mucin bearing epithelia of the original ovarian tumor. Echinococcus implantation during operative manipulation seems to be a related phenomenon.

Turning to the more significant manifestations of epithelial malactivity, we must cite hydatid mole as a recognized forerunner of chorio-epithelioma.

The normal chorionic villi in the early stages of their development consist of an axis or stem of delicate connective tissue covered with epithelium arranged in two layers: the inner layer known as Langhan's layer, the outer layer called the syncytium; this is peculiar and resembles a large elongated multi-nucleated plasmodium, enveloping the villus like a mantle.

In the early stages the connective tissue core of the villus is devoid of blood vessels, the branching cells being separated only by a mucoid intercellular substance. Later the cells become spindle-shaped and the tissue denser and vascularized.

In hydatid mole these villi become changed into transparent, grape-like bodies and look like the vesicles of Taenia echinococcus. One hundred years ago these grape-like bodies were regarded as true hydatids.

In 1827 Madame Boivin and Valpeau held that the disease depended on a change in the chorionic villi. Virchow, in 1853, considered them due to a myxomatous degeneration. This view prevailed until Marchand, in 1895, demonstrated that the essential features of the change depended upon the *epithelium* and not upon the stroma of the villus. He showed conclusively that the epithelial cells underwent *irregular proliferation* and acquired *invasive characters,* penetrating the decidua and even the muscular wall of the uterus.

Such invasive and destructive tendencies of these altered villi have long been recognized, but the significance of the whole process and its relationship to malignancy is only beginning to attract attention.

Chorio-epithelioma is practically the malignant phase of hydatifrom mole, although it may occur independently of this formation.

Even under normal conditions the syncytial epithelia proliferate invade and destroy adjacent tissues, advance into blood vessels to be carried to distant parts of the body.

Palmer Findley states that "for all we are now able to judge, the difference in the behavior of normal syncytium and the malignant type is one of degree in its proliferating and invasive tendencies. We find every possible gradation between the cells of the normal placenta, hydatiform mole, chorio-epithelioma, and transitions from one to the other in the order named. The histological and naked eye appearances are the same in the benign as in the malignant forms and metastatic growths have been known to disappear spontaneously. Here, as elsewhere, we must rely upon the ultimate *clinical* course for the diagnosis of malignancy."

The results of recent investigations have accorded to epithelial cells an inherent potentiality for independent existence and proliferation under certain *activating* influences that are truly marvelous and almost startling in their revelations of biologic possibilities.

Thus Loeb, Fraenkel and others have demonstrated that during certain phases in the periodic formative cycle of the corpus luteum the introduction of any mechanical stimulus into the uterine cavity—be it ovum or foreign body—regularly initiates an active proliferation of the endometrial epithelia, with the resulting formation of a decidua.

This proliferation is ascribed to the activating influence of lutein, an internal ovarian secretion, and according to Pick and many other authoritative investigators lutein hyperproduction is the cause of chorio-epitheliomatous degeneration.

Here, then, is an instance in which a normally reactive proliferation of epithelial structures is perverted into an overgrowth and tumor formation, with the development of malignant manifestations; the same epithelial cells characterize the normal and malignant tissues, being undifferentiable in any, except their ultimate clinical features, and all as the result not of embryologic aberration, not of a germ, but of an altered activating secretion; in other words, of a disturbance in local metabolism.

These findings would tend to substantiate the theory of Bell, of London, who for many years has contended that cancer is the result of a depraved metabolism consequent upon an autointoxication or deficiency or depravation of one or more of the internal secretions, especially of that of the thyroid, and because of this defect restraint on certain cells is removed; in other words, inhibition of their overgrowth is suspended and they run riot, neglecting the functions they are intended to perform and giving themselves up wholly to a debauch of reproduction—becoming a sort of Jukes family, as it were, among cells.

The same line of speculation and hypotheses led to the practice of double oophorectomy for breast cancers and, although some very remarkable retrocessions of growth were observed, ultimate recurrences discouraged the continuance of the operation.

With barely time to realize the deep significance of the lutein phenomenon, we were again startled by Carrel and Burrows' exhibition of tissues and tumors, normal and abnormal, benign and malignant, all placidly growing side by side under the same environment in ordinary glass-test tubes, epithelium of every lineament, of good and bad repute, living, thriving, multiplying and exercising the profoundest vital functions.

Here is the call to shake the dust out of our theories, and Sir Henry Butlin in his Hunterian lectures conceives the cancer epithelium as a tissue cell, transformed into an independent entity—a cell "endowed with that wondrous gift which no man has seen and which no man can understand—the gift of life." It has become a new thing, a new creation. The cell into which has thus been breathed the breath of life now becomes a veritable Frankenstein creation, warring to the death against the tissues from which it sprang. But what is it? In seceding from the metazoan organism, has it returned to the primordeal protozoa, in one bound leaping backward over the evolutionary chasm of eons, or is it a being of a new order?

Butlin would place the new creature among the protozoa, for its resemblance to such is, he holds, very strong.

Like these the cancer epithelia spread in the interstices of the tissues in which they first appear.

They are conveyed to the blood and lymph, they are deposited in distant tissues and organs, and they show the same predilection for certain tissues and organs as do the parasitic protozoa.

They live like these parasites almost entirely by osmosis; sometimes existing for long periods in the body of the host without sign of their presence and perhaps in a state of lethargy until their vigor and activity are stirred by a lowering of the resistance in the host or by some change in the part in which they hibernated, rendering it more favorable to their necessities.

All this is beautiful and impressive, but, unfortunately, the biologist will recognize no such nondescript, and, as Butlin himself laments, "they will have none of it." He consequently created a new order and a new name, calling this transformed epithelium "Unicellula cancri."

This cancer cell is thus *itself* the parasite, the neoplasm is not a tumor developed in response to the stimulation of an extrinsic protozoan or bacterium, but is itself an agglameration or colony of parasites.

The cell being derived from a specialized tissue can produce only its like. Butlin thus explains the various forms of carcinoma and of sarcoma and accounts for their remaining distinct both in the original and metastatic formations.

Butlin claims that his theory explains more of the phenomena of malignancy than any other and that it fulfils the postulates of Koch, with the possible exception of the one demanding that "the cell be isolable and studiable apart from the disease." Here he leans strongly upon the work of Carrel and Burrows, who, as we have seen, succeeded in cultivating cancer cells in artificial media.

If at this present juncture we were to summarize all the evidence of established facts and ask again: *"What* is cancer, *whence* comes cancer and *why* is cancer?" the only possible reply would be embodied in the one term—epithelium.

The first and last steps in the elucidation of any complex problem by modern science is the reduction of that problem to its simplest forms, the analysis of its fundamental primary elements and the synthetic reproduction of all its complexities.

What is epithelium? The unicellular prototype in the highest order of life; the primordeal progenitor of our whole organism; the lowest physiological, anatomical and histological unit in the biological scale known today.

"Omnis cellula e cellula:
Omnis nucleus e nucleo."

Here lies the boundary of all human knowledge; here reigns Isis, shrouding the imponderable riddle of life's beginnings.

"Da steh ich nun, ich armer Thor—
Und bin so klug als wie zuvor."

THYMUS DISEASE IN CHILDREN

WITH REPORT OF A CASE OF THYMUS-HYPERTROPHY

By HERMAN B. SHEFFIELD
New York

We have yet a great deal to learn about the status of the thymus gland in the human economy. Though fully dispensable in adult life, it is essential to the growth and development of the child. The function of the thymus is more or less dependent upon and in part regulated by that of the thyroid, and *vice versa*. Thus, there is less need of thymus when the thyroid is gone, and similarly less thyroid suffices when the thymus is removed. Moreover in cases of hypertrophy of the thymus, where the elaboration of its secretion is excessive, removal of the thyroid is usually followed by sudden death, which shows plainly that in the absence of the regulating power of the thyroid an excess of thymus secretion is destructive to the human economy. Experiments on dogs by H. Close* have shown that about two weeks after total removal of the thymus gland a "stadium adipositas" of from two to three months' duration develops, followed by loss in weight, general bodily weakness, frailty of the bones, arrest of growth in spite of ravenous appetite, and frequently spontaneous fractures. This cachectic state, or *cachexia thymopriva* is accompanied by *"idiotia thymopriva"* with a terminal *"coma thymicum."* Postmortem examination discloses signs of rachitis, osteomalacia and osteoporosis, the lime salts content being reduced to about half its normal percentage, although the ratio of the lime salts was unaltered. According to the same author the deficiency in undissolved lime salts is dependent upon an increased acid action owing to the failure of the thymus to inhibit the formation of acids in the organism or to neutralize or mask an excess thereof. This "hypothetic" nucleinic acid intoxication, he believes, produces in growing bone defective construction, rachitis with abnormal softness and flexibility, while in fully formed bone increased destruction, osteomalacia, and osteoporosis with abnormal fragility.

---

*Brooks and Langerhans Text-Book of Pathology

With these laboratory observations in view we are enabled to obtain a clearer conception of the manifestations accruing from the effects of thymus disease or congenital abnormality, be it hypoplasia or hyperplasia of the thymus.

Thymus disease is variously attributed to lung and heart disease, pertussis, rubeola, diphtheria, scarlatina, asphyxia neonatorum, tuberculous and syphilitic processes, and malignant growths, but excepting the last three etiologic factors, the manifestations produced by the other causes are undoubtedly only transient in character. The same is true of atrophy of the thymus associated with protracted malnutrition. Indeed, it has been shown that the atrophied gland in emaciation is often regenerated on restoration of the body weight. I firmly believe, therefore, that genuine hypo- or hyper-plasia of the thymus is due either to a primary congenital anomaly of the thymus, or develops later secondarily to alterations in the thyroid or other lymphatic gland.

Whatever the cause, the clinical syndromes arising from the effects of hypo- or hyper-plasia of the thymus are not as difficult of recognition as was formerly believed to be. As already stated the functions of the thymus and thyroid glands are closely correlated, so that hypoplasia of the thymus by inducing also a reciprocal diminution in the thyroid secretion produces not only an arrest of growth and frailty of the bones and general debility, but also lowered mental capacity as examplified in infantilism, more especially the Brissaud and Lorrains types.

The clinical signs of hyperplasia differ with the degree of the thymic enlargement and the functional activity of the thymus. Not every case of thymus hypertrophy is necessarily associated with increased function of the gland. The latter may, for example, owe its enlargement to a growth destroying the medullary substance of the gland, and thus be incapacitated rather than hyperactivated. Even where the thymus secretion is excessive the symptoms engendered vary greatly. By corresponding increased activity of the thyroid the clinical picture may be limited to that observed in hyperthyroidism, i. e., tachycardia, insomnia, change in disposition, abnormal perspiration, and often also a mild degree of exophthalmos and possibly struma.

In another group of cases hyperplasia of the thymus careful examination of the child fails to reveal any definite manifestations of a pathologic entity, except possibly a few signs of rachitis or "scrofulosis," and this notwithstanding, death may occur without any apparent reason as the result of slight causes (e. g., serum injection, narcosis, slight operations, etc.) which produce psychical excitement or shock and are usually of no consequence in healthy children. This condition is generally spoken of as *"status lymphaticus."* Unfortunately, we have no characteristic symptoms by means of which this condition may be diagnosed during life and nothing characteristic is found at necropsy.

Finally, in another group of cases hyperplasia of the thymus percussion reveals marked dulness over the upper portion of the sternum particularly to the left as low as the second rib and often also to the back between the scapulæ. It is in addition distinguished by the presence of swollen lymph-glands in the lateral lower region of the neck, which may sometimes be seen to continue deeply down between the clavicle and side of the sternum. If the hypertrophy is of long standing we readily detect secondary manifestations, such as dilatation of the veins of the neck, dislocation of the heart, accentuation of auscultatory signs of the heart and lungs and arching and distension of the thorax. The thymus gland may occasionally be felt as an arched elastic swelling in the middle line above the incisura sterni, which may ascend upward to the thyroid gland. Furthermore, the thymus enlargement is distinctly discernible by means of the Röntgen rays. The clinical signs differ, of course, with the degree of mechanical encroachment upon the adjacent structures (thyroid, bloodvessels and nerves, trachea and bronchi) and secondary involvement of the heart and lungs. As a rule, the symptomatology is essentially that of cardiac asthma, so-called, and is generally spoken of as *asthma thymicum.* The child is suffering from a persistent cough and other signs of bronchitis, marked dyspnea, cyanosis or marked pallor of the face and, off and on, with acute turgescence of the enlarged thymus, it is attacked by paroxysms of asphyxia which not rarely terminate fatally. Pott*, who has frequently observed the course of these fatal attacks of asphyxia, describes them as follows: The children bend their heads suddenly backward (which position, by the way, by producing a marked lordosis of the cervical region of the spine and thus increasing the pressure of the thymus against the trachea greatly aggravates the laryngospasm), and make soundless, gasping inspiratory movements. The eyes are turned, the face is blue or black, the cyanotic tongue is impacted between the jaws, the veins of the neck are distended, the hands are clinched, the forearms are pronated and abducted, the legs are stiff and extended, the large toes are abducted and flexed, and the spine is arched strongly backward. The pulse, heart's action, and heart-sounds cease immediately with the onset of the paroxysm, and after a few futile respiratory efforts the face turns ashy in color and in a minute or two the child is a corpse. Pott believes to have felt the vocal cords snugly together in the median line. He nevertheless maintains that death is caused by heart-failure and not by closure of the glottis, for on two occasions he performed immediate tracheotomy by *one* incision without any relief. This view is not shared by all observers, and Biedert, for example, is of the opinion that closure of the glottis, through suffocation stasis in the heart and thymus, might be responsible for the onset of such an attack and its grave result. Of course, not all attacks terminate immediately fatally. Some time ago I had the opportunity, for several weeks, to watch a five-months-old infant afflicted with an apparently congenital hyperplasia of the thymus. The family history was negative as regards syphilis and tuberculosis. The father, a French artist, was in perfect health, the mother was subject to arthritis with slight valvular heart disease. The older child, who is now ten years old, was for four months suffering from spasmodic pyloric stenosis. The infant under consideration weighed about five pounds at birth and failed to gain though breast-fed for the first three months of its life. It was noticed immediately after birth that he was very pale and cyanotic, had some difficulty in breathing, coughed a little and had a husky cry. Gradually these symptoms became worse and attacks of bronchitis with marked dyspnea set in, during which the baby would repeatedly be seized by convulsions with loss of consciousness. The family physician diagnosed it as asthma, while a very learned pediatrist decided that they were dealing with spasmus glottidis supervening faulty feeding. The child's condition got gradually worse, notwithstanding the correction of diet and administration of antispasmodics, etc. When I saw it the clinical picture resembled pulmonary edema, but there were no signs of primary heart or kidney disease and the history of the case and the distinct hoarseness certainly pointed to some form of immediate obstruction in the upper respiratory tract—most probably primary, nay congenital, since it was manifested at birth. There was no difficulty to exclude asthma, as this disease is invariably associated with remissions and would at least temporarily yield to anti-asthmatic remedies which in this case were given a fair trial. Neither did the diagnosis of spasmus glottidis appeal to me. Here the infant is free from dyspnea, etc. between each paroxysm, and certainly does not get worse on a suitable diet. Going over the case more carefully I detected undue dulness over the upper portion of the sternum, marked dilatation of the veins in the neck, fulness of the neck over the interclavicular notch—all signs and symptoms which were quite suggestive of thymus hypertrophy. I therefore suggested a Röntgen-ray examination, which promptly confirmed my diagnosis. The infant was in too delicate a condition to undergo thymectomy, and his parents—rightly—hesitated to consent to this procedure. The infant was soon relieved of its agony during a paroxysm of asphyxia.

---

*Graetzer and Sheffield Practical Pediatrics, p. 296.

THE ROENTGEN RAYS IN THE DIAGNOSIS OF GASTRIC ULCER, DUODENAL ULCER AND GASTRIC CANCER

By I. H. LEVY
Professor of Medicine, Syracuse University
Syracuse, N. Y.

It is just twenty-one years since Professor Röntgen announced to the scientific world the discovery of the mysterious rays, which for want of a better name have been called "X-rays." While at once of tremendous advantage to the surgeon, to the internist they were of comparatively little value until more recently. Now, owing to the improved tubes, transformers and intensifying screens, they have become an indispensable part of the clinician's armamentarium. So important is the information furnished by the Röntgen rays that no examination can be considered complete without their employment. Just one case illustrating their extreme value when properly interpreted.

When in Vienna in 1912 working with Holzknecht and Haudek, a patient was sent in from one of the medical clinics that had been in the hospital for eighteen days, and despite the most careful clinical examination and laboratory tests, no positive diagnosis could be made. He was running a temperature and looked decidedly toxic. As he was gradually getting worse, and his condition was considered desperate, as a last resort it was decided to have him X-rayed. He was given the usual bismuth meal, and examined in the routine way. It was not until the twenty-four hour observation that anything of any consequence was discovered, and then it was found that all the bismuth was massed close to the diaphragm on the left side, in what appeared to be coils of small intestine. A dense elongated shadow resembling a large bullet was also seen in the left chest. At the end of forty-eight and seventy-two hours the findings were exactly the same. Holzknecht, who made the examination, interpreted the findings as follows: The man attempted to commit suicide by shooting himself in the left side. The bullet penetrated the diaphragm, the small intestine entered through the opening, producing a hernia of the diaphragm. He was sent back with this diagnosis. On closer inspection of the left chest the clinician then found a small recently healed wound. Autopsy some days later confirmed the Röntgen diagnosis. In defense of the clinician, it may be said that in Austria attempted suicide is severely punished, hence the patient's motive for withholding the facts.

It cannot, however, be too strongly emphasized that the Röntgen rays should not be invoked as a first aid, nor should its evidence be contrasted with the clinical evidence. The X-ray examination should always be part of the general clinical examination. It is nothing more or less than interior inspection. The fact that it requires special apparatus, and some technical knowledge, in no way removes it from the domain of clinical medicine to a specialty. It is safe to predict that in the future every physician will be trained to interpret both the fluoroscopic and radiographic shadows.

Gastric ulcer.—The Röntgen rays give us valuable aid in the diagnosis of ulcer of the stomach. The signs may result either from anatomical or physiological disturbance. In general it may be said the more chronic the ulcer, and the greater the anatomical deformity resulting therefrom, the more certain the Röntgen diagnosis. The ulcer leading to pyloric obstruction, to hour-glass deformity, to the "Nische," is readily diagnosed. In some acute cases the incisura, the delayed motility due to pylorospasm, and the sensitive spot, are suggestive. In some cases, especially in the very superficial ulcers, the X-ray findings may be negative. Among the anatomical signs, the incisura is sometimes encountered with ulcer. It results from a spasmodic drawing in of the wall of the greater curvature. The incisura, when present, is quite constant. Atropine may, however, relax it. It is also encountered, but more rarely, with ulcer of the duodenum, with gall-bladder disease, with chronic appendicitis, or any other irritation of the gastrointestinal tract.

The chronic ulcer with pyloric obstruction is readily recognized. The stomach is enormously enlarged, and owing to the atony the opaque meal drops to the bottom of the organ. The peristaltic waves are usually shallow, although in some cases exaggerated as the result of hypertrophy of the walls. There is a marked disturbance of the motor function, the bismuth sometimes remaining in the stomach for days. The rest is half-moon shaped, and considerably to the right.

Haudek has called attention to the "Nische" as a symptom of ulcer. It is found in chronic ulcers with a crater, and particularly in ulcers that have penetrated into neighboring organs like the liver or pancreas. When the crater of the ulcer is still within the stomach wall, it shows itself like a small bud projecting outward from the main bismuth shadow. It is usually found on the lesser curvature. When the ulcer has penetrated into the liver or pancreas, forming the so-called "Ulcus Penetrans Callosum" of Haudek, there is a large pocket in which the bismuth settles and remains. Above this there is a layer of fluid surmounted by a gas bubble. These ulcers are associated with hour-glass contraction, and consequently lead to characteristic deformity.

An hour-glass stomach is not infrequently encountered with ulcer, even without the "Nische." The stomach is made up of two pouches, the food first enters the upper pouch, and gradually finds its way through a narrow channel into the lower one. The deformity may be extensive, the connecting channel usually follows the lesser curvature. The diagnosis of hour-glass stomach rests entirely with the röntgenologist, and not with the surgeon. The operating table, with the stomach empty and collapsed, is not the place to make this diagnosis.

Haudek lays great stress on hypomotility as a sign of gastric ulcer. He claims that he always finds it in the florid ulcer. The delayed emptying is not necessarily due to the pyloric obstruction, but to the reflex spasm of the pylorus. While a six-hour rest is frequently encountered with gastric ulcer, it is not so constant that its absence rules out this lesion.

Holzknecht has called attention to "Schnecken" or "snail" form of stomach as significant of ulcer. The pylorus is found higher and more to the left than normal. In conjunction with other symptoms or signs, this form of stomach may be of some diagnostic value, but alone it is of comparatively little.

Duodenal Ulcer.—Ulcer of the duodenum was for a long time considered a rare condition, but we now know that it is very common—much more so than gastric ulcer. It occurs more frequently in males, and its history is very typical. The condition is usually of long standing, sometimes of thirty years or more. It is characterized by periods of remission, sometimes of years' duration. Its cardinal symptoms are epigastric pains beginning usually two or three hours after eating, that is, when the stomach is nearly empty, and relieved by the taking of food or alkalies, hence the so-called hunger pains.

As in gastric ulcer, so in duodenal ulcer, we have two types of signs, anatomic and suggestive. The anatomic signs are first a deposit of bismuth in the crater of the ulcer, which is rarely encountered, and secondly, constant deformity of the cap. The suggestive signs are hyperperistalsis with or without a six hours' rest, hypermotility, pain point, hypertonus and reflex gastro-spasm. There has been considerable discussion as to the relative value of these two sets of symptoms. Cole has been the chief advocate of the former. According to him, constant deformity of the cap is pathognomonic, and in 500 cases he claims that it never was absent.

For its detection he employs serial radiography, usually making 50 plates, and a normally filled cap in any one of them rules out a duodenal ulcer. There can be no further question as to the value of this sign, and in all probability it is the most valuable single finding. However, the large number of plates necessary makes it expensive and time consuming, and limits its practical usefulness. In the majority of the cases what is accomplished by serial radiography can also be by fluoroscopy. Gastric hyperperistalsis is usually encountered with duodenal ulcer. The peristaltic waves appear early, and are deep cutting, dividing the stomach into two or three ball-like segments. As a result of the hyperperistalsis, the opaque meal begins to leave the stomach very early, almost as soon as ingested. In from one-half to one hour frequently more than three-quarters of the meal has already found its way into the intestines. Hyperperistalsis, however, does not necessarily mean hypermotility. Frequently in cases of duodenal ulcer, despite the hyperperistalsis and early hypermotility, there is a marked gastric hypomotility. In fact, the combination of hyperperistalsis and hypomotility usually means duodenal ulcer.

In duodenal ulcer the stomach is usually small and hypertonic. Even with obstruction this is relatively true, strongly contrasting with ulcers at the pylorus with obstruction in which the stomach is enormously dilated. A pain point is usually encountered with duodenal ulcer. On pressing over the bulbus duodeni while fluoroscoping, pain is elicited. This, however, is not always present. Pylorospasm and incisura, or abnormal drawing in of the greater curvature, has also been observed with duodenal ulcer.

Gastric Cancer.—That the Röntgen rays are of decided help in the diagnosis of gastric cancer can no longer be doubted. In the Mayo clinic this method is considered superior to all other methods —no less than 95 per cent. of the cases give positive signs. The onset of cancer is often so insidious, and its progress so gradual that frequently when the patient presents himself to the physician the disease has already advanced so far that we can no longer speak of an early diagnosis.

The case of Professor Mikulicz illustrates this point admirably. He was one of the pioneers in gastrointestinal surgery, and no one knew better than he the signs and symptoms of early cancer of the stomach. Yet when attacked with this disease he allowed it to progress so far that when he consulted his friend, Professor von Eiselberg, and was operated on by him, the growth had advanced to such an extent that nothing more than the exploratory incision was possible. If so skilled a surgeon could not recognize the symptoms of early cancer in himself, what hope is there for the untutored layman?

In these advanced cases, when the diagnosis is made, there is nothing more to be done, as the time has already gone by when even surgery is able to offer any hope of cure. Cancer of the stomach is so common (about one-third of all cancers are located in the stomach) and as its only hope is an early resection, all means which may aid its earlier detection should be taken advantage of.

The principal radiologic sign of cancer of the stomach is the filling defect produced by the growth encroaching on its lumen. There is absence of the bismuth shadow corresponding to the location of the mass. The growth may involve any portion of the organ, nevertheless, the majority are found at the pylorus, and may obliterate the outlet.. An extensive defect in the pars media may produce an hour-glass contraction. The connecting channel in these cases is usually median and irregular in contradistinction to the hour-glass stomach due to ulcer which is quite regular, and the channel follows the lesser curvature. Not all cases of cancer lead to obstruction or dilatation. As a matter of fact, in cancer the stomach is not enormously dilated. Frequently it is found well above the navel. The disease being of short duration, the stomach does not have time to enlarge as it does with ulcer with obstruction, the exception being an old gastric ulcer that is beginning to degenerate. In the colloid or infiltrating cancer, the lumen of the stomach is very small, the pylorus, owing to the growth infiltrating its walls, is like a rigid tube constantly open. As a result the stomach empties itself very rapidly. In no other gastric condition do we find so marked hypermotility and beautifully filled coils of small intestine. The food seems to run directly into the latter.

When the cancer is of the scirrhus type and obstructs the pylorus, there is marked hypomotility. The opaque meal remains in the stomach much beyond the normal period.

The peristalsis in gastric cancer is also as a rule markedly diminished, although exceptions have been observed. When the pylorus is obstructed, antiperistalsis may occur. The study of the peristaltic wave is very important, as it is known that it is interrupted by the involved wall.

But the X-ray tells us more than that a growth is present. It tells us something of the nature of the growth—whether a scirrhus, medullary or contracting carcinoma is present. It also gives the surgeon valuable information as to the possibility of the removal of the growth, its size, relation to the surrounding structures, and frequently saves the patient an operation. In other words, the Röntgen examination takes the place of an exploratory operation. Although in the diagnosis of gastric cancer unfortunately as yet, the Röntgen rays do not always make the early diagnosis possible, yet I believe it may be safely asserted that, if all cases were seen early enough and X-rayed, the prognosis of the surgical treatment of gastric cancer would be much better.

Now a word as to the negative value of the X-ray in the diagnosis of cancer of the stomach. Not all stomach symptoms in later life are due to gastric cancer. In cases even with a suspicious history, if the Röntgen examination shows a complete stomach outline and a normal motility, an operation should not be advised. It is better to request such patients to report for re-examination four or six weeks later, and if at this examination there is also no filling defect, it is practically safe to exclude cancer.

Syphilis of the stomach has been receiving considerable attention recently. That syphilitics have stomach disturbances that are entirely independent of their infection stands to reason. On the other hand, we have the stomach symptoms due entirely to the syphilitic involvement, and a distinction must be made in these two classes of cases. The radiologic signs of syphilis of the stomach are practically the same as that of cancer. In all doubtful cases a Wassermann should be made and in some, even though that is negative, the patient should be given the benefit of the therapeutic test.

In conclusion, I want to reiterate that the Röntgen examination should not be begun until a careful history has been taken and a complete physical and laboratory examination made. And when made, its findings should form part of a diagnostic chain. Should there be any contradiction of evidence, great care must be used in coming to a conclusion. And finally, it must be considered that röntgenology is only a recent development, and as yet we do not know its full possibilities or limitations. The Röntgen ray simply casts shadows and never makes mistakes, but in the interpretation of these shadows röntgenologists will always make mistakes, as it is human to err.

THE TONSIL AS A PORTAL OF INFECTION OTHER THAN RHEUMATIC

By HAROLD HAYS

Assistant Surgeon in Otology, New York Eye and Ear Infirmary; Assistant Otologist, City Hospital; Adjunct Professor of Laryngology, New York Polyclinic School and Hospital
New York

We are still unable to explain the function of the *normal* tonsil; we are unable to state whether there is such an organ. For it is seldom that a tonsil is removed without showing some evidence of infection and a consequent change in its normal histology.

It is evident to any one who has investigated the subject that the position of the tonsils, their anatomical arrangement, etc., predispose them to infection with all kinds of bacteria. The tonsil occupies an "open" position in the throat, i.e., no food or air can find its way either to stomach or lungs without passing first over the surface of these organs. Moreover, each act of swallowing opens up the various crypts in contact with which come the mouth secretions.

Let us examine the gross anatomy or pathology of the tonsil. The tonsil is an oval-shaped body through which run the deep tubes or crypts. In other words, the tonsil encloses a number of spaces lined with a very sensitive epithelium. On cross-section the crypts are readily seen, covered with columnar epithelium, dipping down into the tonsil, sometimes touching the root of the tonsil itself. Sometimes these crypts communicate. Their lining epithelium is very thin, and is immediately in contact with the parenchyma of the tonsil, which is composed mainly of mononuclear cells.[1]

It is readily seen from this arrangement that an enormous absorbing surface is present. Moreover, the crypts are so arranged that whenever any inflammatory reaction takes place the mouth of the crypt has a tendency to become occluded, thus enclosing whatever infectious material lies within the crypt itself. As the tonsil is a non-secretory organ, and has no way of eliminating any foreign substances except through the mouths of the crypts, one can readily see that gradual absorption has to take place through the system. Is it any wonder then that diseased tonsils cause various systemic diseases far away from the local source of infection?

The question now arises as to the way infectious material from the tonsils enters the system. Wood,[2] of Philadelphia, and Jonathan Wright,[3] of New York, have shown that the epithelium of the tonsil has the property of carrying foreign matter into the depths of the crypts, and that such material in many instances traverses the cryptal lining, and that offending material is destroyed or absorbed by the numerous lymphocytes of the parenchyma. Such investigations have also shown the relation of the tonsil to the lymphatics of the neck, particularly to the so-called tonsillar "gland," lying just anterior to the sterno-cleido-mastoid muscle, below the angle of the jaw. This node is in direct communication with the superficial and deep cervical glands, which often become enlarged at the time of a tonsillar infection.[4] Wood has also proved that there is a definite lymphatic connection between the diseased tonsil, particularly the tuberculous one, through the deep cervical lymphatics to a gland at the apex of the pleura, situated just above the clavicle, the infection being carried by this route into the lungs.[5]

That the tonsil may be the seat of infection (particularly rheumatism, which we shall exclude in this paper) may be readily understood from a study of the gross pathology of the recently removed tonsil. A few years ago a young woman called upon me stating that she had an acute tonsillitis every other day. Such a state of affairs seemed impossible. Yet one day she would present a pair of "clean" tonsils and the next day she would show tonsils with multiple cryptal infection. I decided to remove the offending organs, which was readily done. Cross-section showed that each tonsil was a large abscess cavity filled with thick, creamy pus. Not only had this young woman suffered from a beginning progressive deafness, but she had intestinal disturbances which entirely disappeared after the tonsils were removed.

It is a well-known fact that any local pus cavity may give rise to symptoms elsewhere in the body. And more and more is it recognized that the diseased tonsil is responsible for a variety of ailments, proved by the fact that when the tonsils are removed the trouble disappears. To enumerate these various ailments would force us to tabulate a great number of diseases—appendicitis, pneumonia, nephritis, pyelitis, tuberculosis, anemia, bronchitis, asthma, gastroenteritis, general sapremia and septicemia, etc.

That albuminuria is frequently associated with tonsillar disease is evident from the researches of H. Kosokabe,[6] who shows that the fever is not the cause of the albuminuria, because albumin was found when no fever was present. This writer claims that albuminuria was present in 75 per cent. of his cases. This opinion is corroborated by Bains and Campbell.[7] Within the past year I was consulted about a young man who had a high percentage of albumin in his urine. His tonsils were badly infected and I advised their removal as soon as his physical condition warranted my doing so. Immediately after the tonsillectomy, the albumin increased, no doubt due to the entrance of infectious material into the system at the time of the operation; but this gradually disappeared, and today he is perfectly well.

Thiesen[8] reports seven cases of acute thyroiditis following tonsillitis. Two cases developed well-marked goiter, and two others marked hyperthyroidism.

Taylor[9] reports four cases of general infection following infection of the tonsils. These infections were: (1) a suppurative arthritis of both elbows; (2) a streptococcic infection of the ovaries and peritoneum, causing death; (3) epiphysitis of the hip in a child five years of age; (4) an infection of the leg muscles. Finder[10] claims that the tonsil infection may result in general sepsis, cerebrospinal meningitis and acute anterior poliomyelitis. In the recent epidemic of infantile paralysis sufficient evidence has been obtained that the infection frequently finds its way into the system through the tonsil. This assertion is substantiated by the recent researches of Rosenow.[11] Prouty[12] cites a case of orchitis following tonsillitis.

It is evident from the foregoing data that infected tonsils are responsible for many unexplainable diseases—at least unexplainable as far as the source of infection is concerned. It is not customary to take cultures of the blood at the time that any acute infection of the tonsils is present; yet there is little doubt that many a patient with an acute tonsillitis has a general systemic infection rather than a toxemia. I believe that the bactericidal power of the blood takes care of many of these infections and, therefore, many blood cultures under these circumstances would prove negative. Nevertheless, there is sufficient indirect evidence that bacterial invasion of the blood does take place by way of the tonsils. Every once in a while a case presents itself giving indefinite systemic symptoms similar to those I observed in a physician at one time, who for a period of six months after an acute tonsillitis ran an irregular temperature with loss of flesh and strength.

Just as in the rheumatic sequelæ of tonsillar infection, the other complicating diseases are caused by a bacterial invasion of the bloodstream (except when there is direct extension through the lymphatics of the neck). Wherever an infected embolus finds lodgement, in that place will occur a lesion which can be directly traced to the tonsil. On this assumption an acute attack of appendicitis, after tonsillar infection, is readily explained. So also may be accounted for the complicating lesions such as architis, pyelitis and pneumonia.

When one considers the gratifying results following properly performed tonsillectomy, one cannot help feeling that diseased tonsils are responsible for many ills which are otherwise unexplainable. Whether the lowering of resistance is due to a bacterial invasion or to a general sapremia means little to the patient. What does interest him, however, is the knowledge that after the removal of these organs he is transferred from the domain of the semi-invalid to the enviable realm of the healthy human being.

REFERENCES

1. Harold Hays—Acute Tonsillitis and its Treatment. Med. Rec., May 13, 1911.
2. George B. Wood—Annals of Otology, Rhinology, and Laryngology, March, 1909.
3. Jonathan Wright—Laryngoscope, May, 1909.

4. Harold Hays—Indications for the Removal of the Faucial Tonsils, July 13, 1912.
5. George B. Wood—Annals of Otology, Rhinology, and Laryngology, Dec., 1908.
6. H. Kosokabe—Arch. für Laryngologie und Rhinologie, Volume XXVII, No. 3, page 421.
7. Bain and Campbell—Amer. Med., June, 1913.
8. Thiesen—Albany Medical Annals, 1913, Volume XXXIV.
9. Taylor—Annals of Surgery, June, 1912.
10. Finder—Med. Clinic, December 10, 1911.
11. Rosenow—Jour. A. M. A., Oct., 1916.
12. Prouty—Jour. A. M. A., 1912, Volume LVIII.

THE DIAGNOSTICS OF SOME ABDOMINAL DISEASES OF ORTHOPEDIC ORIGIN

BY SIGMUND EPSTEIN

Orthopedic Assistant, Mount Sinai Hospital Dispensary; Orthopedic Chief, German Poliklinik
New York

The paradoxical instances of abdominal conditions in which an orthopedist is called in to diagnose and treat are few; it is a far cry from abdominal to bone disease. However, medical and surgical diseases occasionally require differentiation from the abnormalities that are commonly known as orthopedic defects. In the upper abdomen, gastric affections are the most frequently encountered, and thus are sure to be thought of before the rarer forms of spinal column disease that may simulate them. Therefore, many cases of pain in the pit of the stomach, really due to a carious spine, are overlooked. Besides the boss and rigidity of the spine in the dorsal region, these cases may be suspected when the patient is a child, when there are night cries, and when the symptoms are relieved by rest in the reclining posture only. In an adult, a sign of value is the jarring sign—severe pain in the back when the subject comes down heavily on his heels.

An epigastric mass may be a cold abscess of the abdominal wall, secondary to disease of a rib.

Herpes Zoster may require differentiation from an herpetic abdominal eruption, sometimes seen in the early stages of spondylitis deformans; spinal stiffness is very marked here; there is severe backache on motion that rotates the affected spinal joints, and the patient is beyond middle age.

Lewis A. Conner has reported several cases of acute gastric dilatation in kyphotic patients, in which after operations, there was sudden and grave obstruction with fatal ending; the spinal kink evidently interfering, mechanically, with normal function of the stomach and duodenum, after recovery from anesthesia. Such may be expected after any operation in a severely hunchbacked individual, when in poor condition; and especially when extensive abdominal repair has been done.

The recent epidemic of poliomyelitis that visited the Atlantic seaboard has been unusual; the sequelæ, the high degree of infectiousness, the attitude of the public, and of the press—all make very interesting reading. But the large number of children left with abdominal palsies will more than occasionally puzzle observers not on the lookout for such cases. When unilateral, the large oblique muscles stretch and relax, giving the impression of an abdominal mass, although it may seem diffuse. When bilateral, the abdomen seems extremely pendulous. I have seen one case that was so distressing to the mother that it was many years before she was able personally to supervise the bathing of the child. The abdominal recti alone are rarely affected. An epigastric hernia is differentiated by the sac, the limited extent of bowel *protrusion*, and by being reducible. Moving from one side to the other, or sitting up, will permit the diagnosis to be made, especially when other trunk or limb palsy exists. See Figs. 1 and 2. On account of the evanescence of the symptoms of disease during the acute stages, many of these cases were formerly overlooked; the histories, therefore, are unreliable.

Percussion and palpation will enable one to exclude tumor, fluid, etc. The frequent occurrence of psoas abscess in lumbar spinal and sacro-iliac caries marks this lesion the one important item in the differential diagnosis of abdominal tumors; the direct diagnosis is, of course, made on finding a limp (psoas contraction), a gibbus in the lumbar region, spinal rigidity (especially in flexion or extension) and an X-ray showing the presence of vertebral collapse or partial destruction. It will be incumbent, however, on the exam-

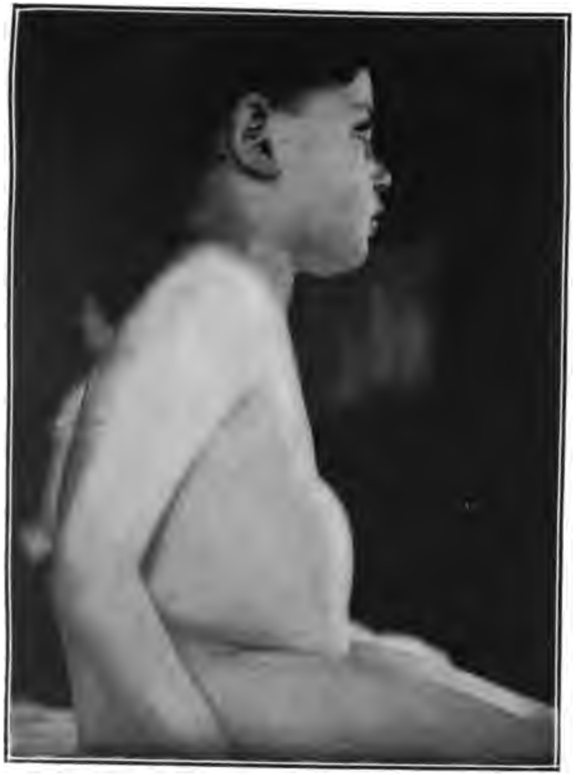


FIG. 1.

Abdominal Paralysis as the only Sequel of Acute Poliomyelitis

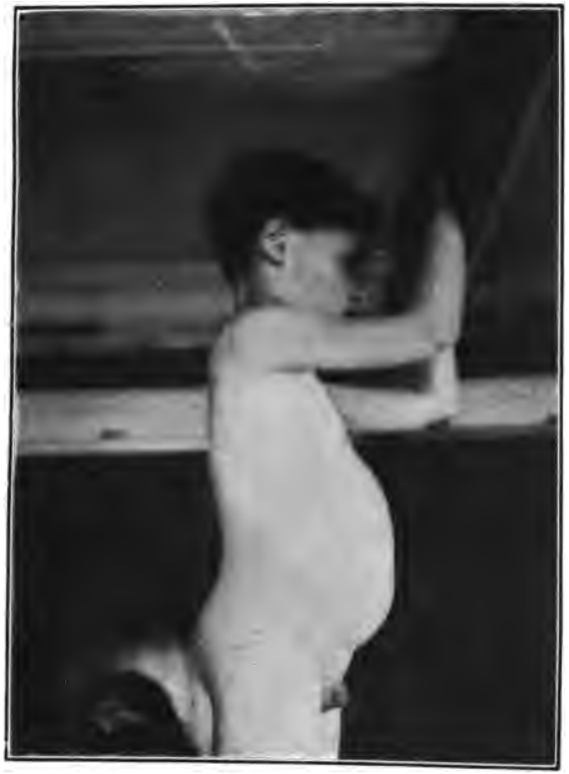


FIG. 2.

Abdominal Paralysis as the only Sequel of Acute Poliomyelitis, 1916 Epidemic.

THE DIAGNOSTICS OF SOME ABDOMINAL DISEASES OF ORTHOPEDIC ORIGIN

By Sigmund Epstein

iner to exclude other intra-abdominal conditions. A psoas abscess is an immovable tumor, felt behind the plane of the intestinal mass; it may be sausage shaped, if it is extensive enough to project beyond Poupart's ligament; the spinal abscess is more or less elastic, depending on its tension (less if there has been an antecedent aspiration). A psoas abscess is chronic, and unless infected, is not accompanied with high temperature. If the patient is laid on his face, the thigh cannot be extended beyond the plane of the trunk on the affected side. Percussion elicits dulness.

Tumors of the bowel, stones in the bowel, are movable: not so smooth, as a rule, as a psoas abscess; fluctuation cannot be obtained. There is different consistency from that presented by a mass of pus bound down by the fascia covering the psoas muscle. A floating kidney is readily movable, and its position is changed during respiration; its very position is the best criterion for its recognition. Affections of the pelvic organs should be readily differentiated by appropriate rectal or vaginal search. An appendiceal abscess is accompanied by abdominal rigidity, leukocytosis, high temperature, and signs of acute disease. I have occasionally been called upon to decide whether or not a mass, in the umbilical region, that was alleged by the patient to be painful, was a projecting vertebra. In a thin individual, this is easy to adjudicate; but in obese young or old subjects there may be difficulty. There may be scoliosis, bringing the sacral promontory a little to one side. The position in which the patient lies will aid to clear up any uncertainty. With perfect relaxation, and legs drawn well up on the flexed thighs, one can follow the well defined line of the promontory, when she lies on the side. By this maneuver, the lumbar lordosis straightens out, and normal relations make themselves felt promptly.

An inguinal hernia may be excluded by the impulse on coughing, the situation of the enlargement, its feel, and its descent into the external genitals. Percussion yields no dulness, and the iliac fossæ are not filled with pus, but I have seen many cases of hernia in kyphotic children. I well remember one child who entered an institution where both affections are treated exclusively. A short time after his convalescence from a hernia operation, a limp and kyphosis were noted, but the course was mild and short, owing to a vigorous constitution. Doubtless an instance of institutional infection. Retroperitoneal abscess is often encountered by the orthopedist, on account of the early limp; the attending physician detects a restricted range of motion in the hip, and coxitis is suspected. But the other motions of the hip (abduction, rotation, etc.) are not interfered with; we are in the presence of acute disease, with rapid pulse, and high temperature, likewise leukocytosis.

A classical case of intussusception does not present any difficulty in separating from a psoas abscess, because it is so often an acute affair; because there are signs of obstruction, the tenesmus and bloody stools help to guide one. Yet we find a sausage-shaped tumor in the iliac fossa in both. I have recently had a case of a boy of seven, who had been doing nicely with his lumbar Pott's disease, when he suddenly developed vomiting and bloody stools; there were rigidity and tenderness in the right iliac fossa, and a mass in the rectum. Operation was averted by the skilful use of enemata and hot baths, in the hands of a colleague.

Sarcoma or osteoma of the iliac bones, although rare, can readily simulate psoas abscess. They usually begin in the flaring portion of the ilium, the patients are usually older than the subjects of spinal caries, the increase in dimension is upward rather than toward the Poupart's ligament, the consistence and shape are different (harder, nodular, and more circular), and there are inguinal adenopathies.

Perinephritic abscesses are seen by the orthopedic clinicians quite often. The patients are sent to us because of their complaint of pain in the back, radiating to the hips; occasionally their presenting a saphenous phlebitis, or their abnormal attitude to one side, is the means of their being sent to the cripple department. Until the case is carefully worked over, the diagnosis is not made with facility; the aspirating needle is the best guide; there will be noted the hematological and thermal findings of infection. The induration or the pus-infiltration is between the last rib and the iliac crest. The possibility of a causative spinal caries infecting the kidney secondarily must not be overlooked.

I could recount the commoner lesions that are diagnosed where Pott's disease really exists, and preach a sermon thereon, but plagiarism is an avoidable sin. Therefore this somewhat random selection of parallels that I have met. In practice, orthopedic aid is invoked when the text-books on diagnosis are unequal to the task of answering the question: When is a belly-ache an orthopedic disease?

CLINICAL DIAGNOSTICS OF DISEASES OF THE HEART

By EDWARD E. CORNWALL

Attending Physician, Williamsburgh and Norwegian Hospitals; Consulting Physician, Bethany Deaconesses Hospital

Brooklyn-New York

(Continued from page 267)

EXOCARDIAL MURMURS

The adventitious sounds arising outside the heart which are produced by the cardiac contractions include pericardial and pleuropericardial friction sounds, velous and arterial murmurs, and cardiopulmonary bruits.

The *pericardial friction sound* is produced by movements of the heart in the pericardial sac when the opposed surfaces of the latter are roughened by inflammatory exudate. This sound may be soft or harsh. In its various qualities it has been likened to the sounds produced by the rubbing together of two pieces of tissue paper, the drawing of a piece of sandpaper lightly across the palm while the back of the hand is held against the ear, and the creaking of new leather. It has usually, though not always, a to-and-fro rhythm, which, however, does not coincide as closely with the rhythm of the regular heart sounds as do endocardial murmurs; and this lack of correspondence may produce the effect of a quadruple rhythm. The sound is extremely superficial, seeming to be close to the ear, and in this respect it differs from endocardial murmurs, which seem of deeper origin. It is usually increased by the erect, and diminished by the recumbent position. It is sometimes increased and sometimes diminished by respiration, but does not show regularly a respiratory rhythm. It is strictly limited to the precordial area. It is first heard most frequently over the base of the heart, and less often over the left border, the apex, and the lower end of the sternum. It usually extends to the apex if it begins at the base, at the same time disappearing at the base or becoming general over the precordium. It is almost always pathognomonic of pericarditis with fibrinous exudate, though it had been observed in non-inflammatory dryness of the pericardium and ecchymoses in its serous membrane, when the heart's action is increased in hypertrophy.

The *pleuro-pericardial friction sound* is produced by friction between inflamed mediastinal pleura and opposed pericardial surface, and can be differentiated from the true pericardial friction sound by the fact that the former has both a cardiac and respiratory rhythm, while the latter has only a cardiac rhythm.

Arterial bruits are most commonly heard in atheroma and aneurism of the aorta. The murmur produced in aortic atheroma is systolic in time and usually softer in character than that produced by aortic stenosis. It can be further differentiated from the murmur of aortic stenosis by the absence of left ventricular hypertrophy, diffuse apex beat, and the other signs and symptoms that go with that valvular lesion. The systolic murmur of aortic aneurism is usually louder than that heard in atheroma, and may be audible over a large area. It can be differentiated from the murmur of aortic stenosis by the other signs of aneurism. The diastolic murmur of aortic aneurism can be differentiated from that of aortic incompetence in the same way, and particularly by the fact that the aortic second sound is clearly audible and may even be accentuated.

Venous bruits are usually heard over the jugular and subclavian veins. They are systolic in time, and possibly are caused by inequalities in the caliber of the veins. They are observed in anemia and nervous conditions.

Cardio-pulmonary murmurs are caused by pressure of the contracting heart against a piece of aerated lung pinched between it and the anterior chest wall. They vary widely in intensity, quality and pitch with the varying physical conditions. They may be bubbling or splashing in character, or so rasping as to simulate a pericardial friction sound. They may be loud enough to be heard several inches from the chest.

THE PULSE—CARDIAC ARRHYTHMIAS

The examination of the pulse for diagnostic purposes was practiced in ancient times, and it remains now an important as well as convenient diagnostic procedure. The pulse is an index of the action of the heart; not an absolute one, indeed, because in certain conditions some of the ventricular contractions are too weak to produce a perceptible wave in the arteries; but a determinative one in most conditions as regards the rate and rhythm of the heart's action, and, also, to a certain extent, its quality. Recently graphic methods of examination with the polygraph and electrocardiograph have added much to our knowledge of the action of the heart in health and disease; but for general clinical purposes, after having learned from the results of investigations with the graphic methods what to look for, it is possible by digital examination of the pulse and inspection of the jugular veins to find out most of what the graphic methods could reveal in the particular case. By digital examination, as Broadbent has said, every important variety of the pulse which the sphygmograph reveals, was recognized, described and named before the Christian era. The established reputation of the pulse as an index of the circulation makes it permissible to combine its discussion with that of the cardiac arrhythmias.

What the pulse is and how it is produced

The pulse is a wave of increased pressure in the arterial system which is started by the ventricular systole. The action of the heart, although the prime factor in its production, is not the only factor; there remain the elasticity of the great arteries and the resistance of the small arteries and capillaries. The heart determines absolutely the rate and rhythm of the pulse, and, subject to qualification by the other two factors, its force. The character of the pulse wave is to a large extent determined by the elasticity of the arteries, which transforms the intermittent jet of the heart into a continuous flow; and by the peripheral resistance, which determines the mean tension of the pulse, modifies to a certain extent its wave, and affects its rate and rhythm by reflex influence on the action of the heart. In estimating the diagnostic significance of pulse findings in diseases of the heart and arteries, it is necessary to keep clearly in mind the part played in their production by each of these factors.

How contractions of the heart are produced

Two kinds of muscle have been demonstrated in the heart, the primitive muscle, whose strands extend without break of continuity throughout the entire organ; and the mature muscle, whose strands are not continuous throughout the heart, but are of local distribution. It is the former which possesses the power of initiating contractions. The primitive muscle, besides being generally distributed, shows several aggregations of its tissue, of which the most important are: The sinoauricular node, situated at the upper end of the cardiac tube; the auriculo-ventricular node, situated low down in the auricular tissue at the right posterior edge of the septum; and the auriculo-ventricular bundle, or bundle of His, which is a band extending from the auriculo-ventricular node downward into the ventricles. This bundle, apparently, is the only channel of communication between the primitive muscle of the auricles and that of the ventricles. From the fibers of the primitive muscle contraction impulses are communicated to the fibers of the mature muscle.

While impulses to contract may originate in any part of the extensively distributed primitive muscle, they do so most readily and most frequently in the sino-auricular node, which is the place of origin of the regular, normal contractions of the heart, and which, therefore, has been very aptly called the pacemaker of the heart. Originating here, the impulses sweep over the auricles and ventricles in regular order; smothering, so to speak, all impulses which might arise independently in the other nodes or regions of primitive muscle, by taking advantage of the fact that impulses cannot arise in them during the "refractory period," which persists for a certain time after each contraction: before an independent contraction can develop, another impulse from the pacemaker utilizes the recovered contractility.

Besides the property of initiating contractions, the heart muscle has other properties which are called into play in the exercise of its function; viz., irritability, conductivity, contractility and tonicity; which should be borne in mind in order to understand how the regular action of the heart is produced, and to appreciate the significance of abnormalities in its action

Cardiac arrhythmias

Variations from the normal in the contractions of the heart may occur in their time relations and in their force.

Variations in frequency may take place within wide limits in health, but if extreme or permanent, they may have a pathological significance.

In *simple tachycardia* the contractions originate in the sino-auricular node and follow the usual course throughout the heart. Among the conditions which cause increased rapidity of heart action are fever, organic disease of the valves and myocardium, anemia, overexertion, hyperthyroidism, and nervous and mechanical disturbances.

In *paroxysmal tachycardia* the contractions do not necessarily originate in the regular pacemaker, the sino-auricular node, but in some other part of the primitive muscle, and are excited by some abnormal stimulus. The contractions are, in reality, extra systoles, and, possibly in some cases, may be produced by auricular fibrillation. The tachycardia in this syndrome is characterized by a high rate, usually 150, 200, or more per minute, and by a sudden onset and disappearance. The syndrome may also include pallor and distension of the veins, and sometimes a sensation of fluttering or distress in the precordium. If the attack lasts long, there may be enlargement of the liver, edema of the lower extremities and lungs, and dilatation of the heart.

The heart may be abnormally slow in its action as a whole or in parts, but it seems best to restrict the term *bradycardia* to the slow action of the heart as a whole, using the term heart block for those cases in which the ventricle alone contracts slowly on account of a blocking of the passage by which come to it the more rapid impulses originating in the auricles. Bradycardia, as here defined, almost never shows a pulse rate below 40. While it is often a sign of disease, it may be habitual and posses no apparent pathological significance.

The causes of bradycardia belong in two classes, those which act directly on the sino-auricular node to depress its function of stimulus production, and those which come through the vagus nerve. The pacemaker may be depressed by the toxic agents produced by certain bacterial diseases, notably typhoid fever, diphtheria and influenza; by those which develop during convalescence from acute infections; by poisonous substances retained in the blood in jaundice and chronic-nephritis; by the poisons of intestinal putrefaction; and by poisons directly introduced, such as lead and digitalis. The vagus may be stimulated by direct irritation of the pneumogastric center in the medulla, as a result of abnormal conditions in the brain; or stimuli inhibiting the pacemaker may come through the vagus reflexly from abnormal conditions in the abdominal viscera, such as gastric pain and irritation and hepatic or renal colic. Bradycardia may be observed in senile degeneration of the heart.

In *sinus arrythmia* each individual contraction originates in the sino-auricular node, but the intervals between the contractions vary in length. These variations usually affect the diastolic period only, and may correspond with the phases of respiration. They are probably due to influences which come through the vagus nerve. The radial pulse is found constant in volume, though irregular in rhythm. The differential diagnosis must be made from extra systoles, heart block and auricular fibrillation. This form of arrhythmia may be without pathological significance.

Extra systoles are contractions which are due to some other cause than the regular, rhythmic stimulus production of the pacemaker. They may originate in any part of the primitive muscle, and be excited by stimuli in the muscle itself or extrinsic stimuli. Among the extrinsic stimuli may be irritation from patches of degenerated cardiac tissue, increased intracardial pressure, chemical stimuli from faulty metabolism or ingested poisons, and nervous stimuli. Extra systoles are rendered possible by defects in conductivity.

Extra systoles which occur in the ventricles are usually limited to the ventricles. The contraction occurs a little before the regular time, and the ventricle, being in its refractory period when the regular stimulus comes from above, misses it; and consequently the period until the next regular contraction is due, which is called the compensatory period, is longer than the period between the regular beats.

Auricular extra systoles present much the same features as ventricular extra systoles, but with them the compensatory period is usually incomplete.

Subjective sensations may be entirely absent, or there may be a feeling as of a thump in the chest when the extra systole occurs.

When extra systoles occur regularly with every second cardiac contraction the pulsus bigeminus is produced: a strong beat, a short pause, a weak beat, a long pause. Similarly, the pulsus trigeminus is produced. The pulsus bigeminus can be differentiated from the pulsus alternans by the inequality in the intervals between its beats.

The significance of extra systoles is not always clear. They may be found in persons who show no other evidences of heart disease.

In *auricular fibrillation* impulses to contract arise in many parts of the auricle simultaneously and in rapid succession, and the auricle contracts "piecemeal." Of these multitudinous impulses only a comparatively small number are able to reach the ventricle at the time when recovery from its refractory period makes it receptive. The ventricle responds to as many of them as it can, but necessarily in an irregular manner, and the pulse is absolutely irregular both in time and force.

Occasionally the presence or inception of auricular fibrillation is attended with a sensation of fluttering in the heart.

This condition is commonly found in the later stages of mitral stenosis, and may occur in any case of myocardial degeneration, especially if it involves the auricles. Its significance is grave, although patients may live with it for some years.

Heart block is a condition in which there is interference with the conduction of contraction stimuli from one part of the heart to another. The form in which it most commonly appears is auriculo-ventricular heart block, due to disease or injury of the bundle of His. This blocking of the passage between the auricles and ventricles may be complete or only partial. In complete heart block the upper and lower chambers of the heart beat independently of each other; the auricles contracting regularly in response to impulses coming from the pacemaker, and the ventricles contracting in response to local stimuli. As the stimuli originating in the ventricles develop more slowly than those which originate in the sino-auricular node, the ventricular contractions are slower than those of the auricles. In partial heart block there is depression of the conductivity of the bundle of His, but not complete dissociation of all its fibers; and some of the impulses from the auricles are able to get through to the ventricles and cause contractions of the latter. When these impulses fail to come through, the ventricles drop a beat. These dropped beats may occur occasionally, or they may happen with every second or third auricular contraction. If the periods of dissociation are prolonged, contractions may originate in the ventricles as in complete heart block.

The pulse in complete heart block represents the ventricular contractions, and its rate is usually below 40, and may be much slower than that. It is usually regular. In partial block the pulse may be regular or irregular, and is, as a rule, faster than in complete block.

The pulsations of the jugular veins, if they can be made out, are found to be more rapid than the radial pulse.

The symptoms of heart block depend largely on the amount of circulatory incompetence, and may include precordial sensations, even to the extent of angina, and cerebral symptoms, even to the extent of unconsciousness. When the latter occurs, the Stokes-Adams syndrome is seen.

Intraventricular heart block has been described as a rare condition, in which the two ventricles contract independently, with production of hemisystoles; or in which one part of a ventricle contracts independently of the rest. Also, sino-auricular heart block has been described, in which the auricle contracts independently of the sinus, and with a slower rhythm.

Pulsus alternans is a condition in which a weak beat follows a stronger one, the intervals between them being approximately equal. It is probably a sign of depression of contractility. It is found in senile hearts with fibroid degeneration, and in other forms of myocardial degeneration, and in hearts which have undergone exhausting strain. It is not infrequently complicated by extra systoles.

Pulsus parodoxus is a name given to a form of cardiac irregularity in which the pulse diminishes in size or is lost during inspiration. It seems to be without diognostic significance.

Variations in the character of the pulse wave

The pulse wave may vary in duration, in amplitude, in tension and in special points.

The *duration* of the pulse wave is long when a strong ventricular contraction is acting against increased peripheral resistance, and short in opposite conditions. It is long in hypertrophy of the heart, particularly if associated with chronic nephritis or arteriosclerosis. It is short in most cases of myocardial debility, and in aortic incompetence even when the heart is acting strongly.

The *amplitude* of the pulse wave is small in cases of high blood pressure with contracted arteries, and in cases where the output of the blood from the heart is restricted, as in many cases of mitral stenosis and aortic stenosis. It is also small with very feeble action of the heart. It is large in cases with a strongly acting heart and dilated arteries, and in cases of low blood pressure where the heart is acting strongly. It is large in most cases of aortic incompetence and in most cases of hypertrophied heart without valvular lesions.

The *tension* of the pulse, both at the crest of the systolic wave and in the trough of the diastole, is equivalent to the intra-arterial blood pressure, if we allow for the resistance of the arterial walls. As the pulse itself is a wave of increased blood pressure, variations in the tension of the pulse can be discussed most satisfactorily in terms of blood pressure, and such discussions will be taken up in a separate section.

There are certain *special points* of variation in the character of the pulse wave which can be appreciated by the finger, although they are brought out with greater precision by the sphygmograph, which have some diagnostic value.

In the dicrotic pulse the secondary wave reflected from the aortic valve and the elastic walls of the great arteries is exaggerated. It is found in cases with relaxed arteries and low blood pressure, and is a common sign in the later stages of typhoid fever.

In the anacrotic pulse the secondary elevation in the wave is higher than the first. This phenomenon is due to a disturbance of the normal relationship between the pressure of the pulse wave entering the arteries and the resistance of the latter: the elastic recoil of the aorta does not manifest itself. It is seen in sclerosis of the aorta and in aortic stenosis.

The pulsus bisferiens is characterized by a secondary elevation very near to the primary one, and nearly equal to it in height. It is observed most commonly in sclerotic disease of the aorta.

BLOOD PRESSURE

The pressure of the blood in the arteries is not the measure of the ventricular contraction alone, but the product of that and at least one other constant factor, viz., the resistance in the arterial circuit. Both of these prime factors, though constants, are under the influence of variants; and the variants are numerous and complicated. In fact, so flexible is the circulatory apparatus in its responsiveness to the varying demands of health and disease, and so sensitive is it to the agents which modify both the systolic and the vasomotor activity, that the diagnostic significance of variations in the blood pressure is neither as great nor as definite as might be inferred from a superficial consideration of the subject. The wide variations in blood pressure which are observed in health, and the allowances which must be made for the neurotic and other subsurface factors in the patient, which affect the systolic contractions and the varying tonus of the arterial musculature, detract much from the face value of blood pressure findings in diagnosis. Recognition of this fact seems to have led recently to a certain amount of reaction from former enthusiasm as regards the diagnostic value of examinations of the blood pressure; and R. C. Cabot (1915) goes so far as to express the opinion, that very little information of clinical value is obtained from the diastolic pressure; but with that opinion the generality of observers probably will not agree. Notwithstanding the confusion of the subject, there seem to be some diognostic inferences of positive and considerable value which can be made from blood pressure findings.

The significance of blood pressure findings in diseases of the heart can be best understood if they are also considered in their general diagnostic relations.

The pressures alluded to in the following discussion are those which are measured in the arm by the clinical instruments in ordinary use and by auscutatory method.

The three phases of blood pressure and their relation to the efficiency of the circulation

Blood pressure appears in three phases, viz., the systolic pressure, which is the highest pressure observed in the pulse wave, and is the measure of the force of the ventricular contraction modified by the arterial resistance and less important factors; the diastolic pressure which is the lowest pressure observed in the pulse wave between the ventricular contractions, and may be taken as the residual pressure maintained by the tonicity of the arterial system, and which measures the resistance against which the systolic pressure is raised; and the pulse pressure, which is the difference between the systolic and diastolic pressures.

The measure of circulatory efficiency is the quantity of blood which passes through the tissues in a given time. It depends on a number of factors, chief of which are, the quantity and quality of blood delivered to the heart, the force and frequency of the ventricular contractions, and the amount of the peripheral resistance. An exact estimate of the combined, co-ordinated action of these factors is impossible. A very useful, though only partial, index to the efficiency of the circulation, however, is afforded by the systolic and diastolic pressures considered in their numerical relations, that is, the difference between them, which is the pulse pressure, and the relation of the pulse pressure to the pressure scale.

The pulse-pressure tells us the fall in pressure between the heart and arterioles, and is analogous to what is termed a "head" of water in hydrostatics. But this fall, as an index to the circulation, is conditioned to a considerable extent by its place in the pressure scale. A given pulse-pressure in one part of the pressure scale may not have the same value as a factor in the circulation as the same pulse-pressure in another part. For example, a pulse-pressure of 40 mm. Hg. produced by a systolic blood pressure of 200 and a diastolic pressure of 140, will not, naturally, be attended with as efficient a circulation as the same pulse pressure produced by a systolic pressure of 120 and a diastolic pressure of 80. A theoretic conclusion, which receives practical support, is that there is a particular place in the pressure scale for each individual and each particular condition, where a given pulse pressure, other things being equal, will produce the fullest circulation, and which at the same time will be attended with the greatest circulatory flexibility and reserve power; and that pulse pressures placed above or below this ideal place in the pressure scale, in order to produce an adequate circulation, must ordinarily increase in magnitude, either absolutely or relatively; and that such increase means diminution in the flexibility of the circulation and encroachment on the reserve power of the heart. If the diastolic pressure fails to fall in just correspondence with a falling systolic pressure and thereby permits the pulse pressure to become unduly small, clinical symptoms of circulatory insufficiency may appear; and if the systolic pressure fails to rise in just correspondence with a rising diastolic pressure, thereby permitting the pulse-pressure to become unduly small, the same lessening of circulatory efficiency may be brought about. By the term "just correspondence," used in connection with the rising and falling in reference to each other of the systolic and diastolic pressures, is meant an increase of the pulse pressure at least adequate to the bare needs of the circulation.

The determining factor for the pulse-pressure, at least in conditions of disease, seems to be the diastolic more than the systolic pressure. Movements of the diastolic pressure in either direction from its normal place in the pressure scale seem regularly to cause the pulse pressure to increase in size, either absolutely or relatively, if the circulatory balance is preserved. In cases where the systolic pressure rises while the diastolic falls, it is difficult to determine which is chiefly responsible for the increase in pulse-pressure, but it may be inferred that the increase is compensatory for either a functional or a pathological condition. This phenomenon is observed in apparently healthy hearts responding to a transient demand, and also sometimes in cases with pathological conditions which balance each other. A normal pulse-pressure, as well as normal systolic and diastolic pressures may be observed in cases of advanced cardiac disease which show signs of cardiac insufficiency.

Normal blood pressure

Before attempting to interpret the clinical significance of specific variations in the three phases of blood pressure, one should have a conception of the normal character of those phases and of their range in apparent health. Such a conception is not easy to secure. Exact standards of normal blood pressure do not exist; blood pressure conditions in health and disease overlap widely; pressures which are without significance in some conditions may possess such significance if found in others; and a considerable allowance must always be made for individual peculiarities. At best, only approximate figures for normal blood pressure can be given; which, however, may serve as a general guide, due reservation being made for exceptions. It is obvious that the intepretation of blood pressure findings may require the exercise of a highly cultivated clinical judgment. With these cautionary remarks the following figures are offered.

The systolic pressure may range in healthy young adults between 100 and 130, being mostly between 110 and 125. In people of middle age it may range about ten points higher, and in elderly people, nearly twenty points higher. But in young and middle-aged people a systolic pressure of 140 is often observed without apparently indicating serious disease, and elderly people who appear to be in good health often show a systolic pressure no higher than do young or middle-aged adults. In young adults a systolic pressure of 100 may be considered low, as may one of 110 for a middle-aged person, or one of 120 for an elderly person.

The diastolic pressure may range between 60 and 90 in young adults, between 75 and 95 in the middle-aged, and between 80 and 100 in the aged.

The pulse pressure in health is usually not far from 40, and it may vary between 30 and 50 without necessarily possessing pathological significance. A pulse pressure as low as 20 or as high as 60, if persistent, suggests a pathological condition.

Conditions which generally produce elevation of blood pressure

In *chronic nephritis,* particularly chronic interstitial nephritis, the blood pressure is regularly raised, both systolic and diastolic, and the elevation, especially of the diastolic pressure, is more or less proportional to the severity of the kidney lesion. The diastolic pressure may rise to only 100 in mild cases, but in severer ones it rises to 120, 140, 160, and even higher. A high diastolic pressure is characteristic of the blood pressure picture in chronic nephritis. And along with the rise in diastolic pressure goes a rise in the systolic pressure and an increase in the pulse-pressure to meet changed requirements of the circulation. The systolic pressure may rise to 180, 200, 250, 300, and even higher. In order to maintain this compensatory high pressure the left ventricle regularly hypertrophies; and such hypertrophy is as much a part of the pathological basis of the high blood pressure as the nephritis. When this hypertrophy has reached its limit and myocardial degeneration has developed to a notable degree, and there is no more reserve power in the heart, the systolic pressure ceases to rise, and, as the case progresses, it declines. The diastolic pressure at first may fail to decline in correspondence, but finally it does so, and the breaking up of the circulatory apparatus is hastened by the failure of elimination. While the circulatory compensation of the kidney insufficiency is adequate, the pulse-pressure is regularly large. It increases in a more or less geometric ratio with the increase in the diastolic pressure, being about 60 in mild cases, which is nearly within normal limits, and in severer cases, 80, 100 and even more. With a diastolic pressure of 130 to 140, a pulse-pressure adequate to maintain compensation of the kidney insufficiency will usually be found to be not far from 100.

In *arteriosclerosis* the blood pressure changes seem to be less regular and uniform, less obstinately persistent, and less definitely characteristic than in chronic nephritis, and they seem to be more susceptible of modification by other conditions than in the latter disease; although in the latter disease myocardial failure can radically change the blood pressure picture; and arteriosclerosis is usually more or less involved in it, though apparently subordinated. Arteriosclerosis, even when not overshadowed by nephritis, is often so involved with other conditions that the blood pressure picture which it produces is not clear. Moreover, the lesions of arteriosclerosis differ widely in their intrinsic influence on blood pressure according to their character and distribution: some have very little influence, while others, notably lesions affecting the aorta, the coronary arteries, the splanchnic arteries, and the arteries of the brain, may have a great influence. The diastolic pressure is usually raised, but, as a rule, not so much as in nephritis. It is considerably elevated when the blood supply of vital regions is affected. The systolic pressure is raised to produce the adequate pulse-pressure. When myocarditis complicates, as is often the case, the enlargement of the pulse-pressure seems often to be producd in part by lowering of the diastolic pressure, thus securing a relief which does not seem to be available in chronic nephritis. As raisers of the blood pressure, both systolic and diastolic, arteriosclerosis and chronic nephritis stand out prominently, and they are usually the first conditions thought of when persistent elevation of the pressure is found. In cases of high blood pressure due essentially to arteriosclerosis, the entrance of a cardiac factor is as inevitable as in the case of chronic nephritis, and for the same and other reasons: besides exciting hypertrophy to maintain high blood pressure, the sclerotic changes may involve the coronary arteries or their branches, with resulting nutritional and degenerative changes in the myocardium; and they may involve not only the aorta itself, but the valves at the aortic orifice, with production of a valvular defect and its mechanical consequences; both of which conditions may produce their characteristic modifications of the blood pressure picture.

In *chronic lead poisoning* the blood pressure is usually elevated in much the same manner as in arteriosclerosis.

In *apoplexy, cerebral thrombosis, intracranial hemorrhage, brain tumors of rapid growth, and any condition producing increased intracranial pressure,* there may be very great increase of both diastolic and systolic blood pressure, in order to meet the urgent demand to overcome the obstruction which interferes with the delivery of sufficient blood to the brain tissue. In these conditions anemia of the vasomotor center may bring about extreme constriction of the arterioles and a responsive increase in the force of the ventricular contractions.

In *vascular crises,* including some cases of angina pectoris, there may be vasoconstriction and rise of both systolic and diastolic pressures.

As a result of *asphyxia* due to failure of the heart in the later stages of valvular disease, and in other conditions, notably pneumonia, there may be a temporary rise of blood pressures.

The effect of *ether anesthesia* on the blood pressure of patients in good general health, who are operated on for local conditions, seems to be to produce an elevation of ten to twenty points in the systolic pressure which lasts about four days, without much change in the diastolic pressure. This conclusion is based on observations made under the writer's direction on ten such cases.

Conditions which generally produce lowering of blood pressure

In *chronic myocarditis* or any condition of chronic myocardial degeneration the systolic pressure shows a tendency to fall below the ideal for existing conditions, and the diastolic pressure to accommodate itself to the systolic pressure when it can, and the pulse-pressure to enlarge excessively. In uncomplicated myocarditis both systolic and diastolic pressures may be below normal, and the pulse pressure larger than normal. The effects of myocarditis on blood pressure, however, may not be evident for a considerable time, being apparently spent on the reserve power of the heart; when that is exhausted, then the natural effects appear. The lowering of the diastolic pressure in this condition relative to the lowering of the systolic pressure is, in general, an index of the degree of myocardial efficiency, in cases which are not complicated by chronic nephritis and arteriosclerosis. In such cases the blood pressure picture is less distinct, and there appears a tendency on the part of the diastolic pressure to resist adequate compensatory lowering, and the systolic pressure to remain high; and cardiac breakdown to appear early.

The writer has seen a number of elderly people who had obvious myocardial degeneration, but no apparent nephritis or arteriosclerosis, with blood pressures as low as 100 to 120 systolic and 50 to 75 diastolic, who were comparatively free from circulatory disturbances while they led quiet lives. In some of these cases probably the low pressure was due to other causes than myocardial degeneration; among which chronic toxemia of intestinal origin and chronic tobacco poisoning may have had a place.

In *chronic wasting diseases,* in *diarrhea,* in *shock,* and in *hemorrhage,* the blood pressure is regularly lowered.

In the *acute infectious diseases* (excepting meningitis), the blood pressure is usually lowered, and perhaps as much by disturbance of the vasomotor center by the specific toxemia, as by weakening of the myocardium and the arterial walls.

Chronic toxemia of intestinal origin appears to be a factor of considerable importance in producing variation of the blood pressure, and its effects seem to differ in different conditions. With chronic nephritis and perhaps arteriosclerosis, it seems able to raise still further the systolic pressure and perhaps the diastolic also; but in cases in which those conditions are absent or not present in marked degree, its effect seems rather to be a lowering of both systolic and diastolic pressures, and perhaps of the systolic more than the diastolic. A marked variability appears in its action. On the whole, its natural effect seems to be more that of a depressor than an elevator of the blood pressure. The part played by this element in any given blood pressure picture can be estimated approximately by means of the therapeutic test. If the amount of toxemia is diminished by appropriate dietetic treatment, the resulting alterations in the blood pressure picture may show by subtraction the effect on the blood pressure of the toxemia in the given case.

Chronic tobacco poisoning seems to be a frequent cause of low blood pressure, both systolic and diastolic. In cases showing mild anginoid attacks the writer has observed that the diastolic pressure was not lowered in just correspondence with the systolic, resulting in an insufficient pulse-pressure.

In *chronic valvular disease* with failing compensation there may be low blood pressure, both systolic and diastolic but when compensation is good, the blood pressure is usually near the normal, except in the case of aortic incompetence, where the systolic pressure is regularly high and the diastolic low.

General remarks on the clinical significance of blood pressure findings

The following remarks which are based largely on the writer's own observations, are not intended to be taken as dogmatic and conclusive, but only as tentative and suggestive.

A comparatively low systolic pressure, provided the diastolic pressure is sufficiently low to provide an adequate pulse-pressure, does not necessarily mean poor circulation, though it may imply diminished reserve power of the heart.

A comparatively low systolic pressure with a comparatively high diastolic pressure and a consequently small pulse-pressure, may mean myocardial weakness complicating chronic nephritis, arteriosclerosis, chronic toxemia, or arterial spasm.

A low diastolic pressure with a comparatively high systolic pressure and an excessively large pulse-pressure, may mean several things, e.g., a purely functional condition, a compensated aortic incompetence, myocardial degeneration without much arteriosclerosis or nephritis, or vasodilatation from any cause.

The diastolic pressure seems to be more stable than the systolic, and to show less often than the systolic marked variations from its normal without definite patholigical cause; and the systolic pressure seems to accommodate itself to the diastolic more readily than the diastolic to the systolic in conditions of disease. The diastolic pressure seems to indicate the peripheral resistance, which in many cases is determined by definite pathological conditions. Movements of the diastolic pressure beyond the normal range seem to be a sufficient cause for enlargement of the pulse-pressure if an adequate circulation is to be kept up.

Heart stimulant drugs seem to improve the circulation in some cases even when they do not raise the systolic pressure. Vasodilator drugs may improve the circulation by increasing the pulse-pressure. Cardiac depressant drugs may lower the diastolic pressure by weakening the systolic force and thereby calling for an enlargement of the pulse-pressure.

A systolic pressure of 100 or lower, if persistent, suggests disease. A diastolic pressure of 100 or higher, if persistent, suggests disease. A systolic pressure over 140 in a young or middle-aged person, if persistent, suggests disease. It may be possible for a person to live for several hours with a systolic pressure as low as 60, for several days with one of 70, and to walk around without particular symptoms of circulatory distress with one of 90, provided the pulse-pressure is sufficiently large. A pulse-pressure as small as 20 or as large as 60, if persistent, may be pathological.

A fall of systolic pressure in response to an exercise test, immediately or after a short preliminary rise, and a delay in its return to the normal; and also a failure of the pulse-pressure to show a substantial increase in size, suggest myocardial weakness.

Shortly before death partial asphyxiation may cause a sudden, transient rise in the blood pressure.

Toxemias, both acute and chronic, may lower the systolic pressure without much increase, or with diminution, of the pulse pressure. But in most patients with high blood pressure, the taking of a non-putrefactive diet is followed by lowering of the pressure.

Chronic tobacco poisoning seems usually to be attended with low pressure.

Chronic nephritis is regularly attended with high systolic and diastolic pressures, and a large pulse-pressure, which latter continues to be adequate as long as the myocardial reserve power holds out.

Arteriosclerosis, as a rule, seems to produce marked elevation of the blood pressure only when the blood supply of a vital region is affected.

II

SPECIAL DIAGNOSTICS

In the preceding part of this treatise the diagnostic significance of the individual symptoms and signs which point to diseases of the heart was discussed. In this part the subject is dealt with from the other, the special side, and the sign and symptom groups characteristic of particular diseases are described. It is by comparing the signs and symptoms which we find in any particular case with the various signs and symptoms characteristic of the different heart diseases, that we are enabled to make the special or differential diagnosis.

The sign and symptom groups characteristic of the different diseases constitute the clinical pictures of those diseases, and these clinical pictures are not fixed but moving pictures, which change characteristically with the clinical course of each disease. Therefore, in special diagnosis the clinical courses characteristic of the various diseases must be brought into comparison with the clinical course of the patient whose disease we are trying to identify.

The etiology, also, may help us in making the diagnosis.

If the special diagnosis can not be made by a consideration of the etiology, the signs and symptoms and the clinical course, the ultimate recourse is an examination post mortem.

The classification of diseases of the heart which is here followed is, with slight variations, that found in most of the text books, and therefore generally familiar. Not all the cardiac conditions described in the text books are mentioned; and some of the less common and important ones are passed over with scant notice; and in no case is the attempt made to do more than present the most important diagnostic points. Brief pathological allusions are made for the purpose of suggesting the pathological definitions where possible; but the pathological pictures drawn are outline sketches.

PERICARDITIS

Inflammation of the pericardium may be limited to a small portion of its inner surface, but usually it spreads over the greater part or the whole of that surface. It may involve only the tissues of the pericardium, or extend to the myocardium and endocardium, and even the pleura and mediastinum. The inflammatory exudate may consist only of fibrin, or there may be also a serous, serosanguinous, or purulent effusion into the sac. The inflammation may run its course and subside without leaving important anatomical changes, or adhesions may remain which bind the pericardium to the heart or to adjacent structures. Its course and outcome may be greatly modified by coexisting or complicating diseases.

Etiology

The most common cause is rheumatic fever. Rarely pericarditis is observed as the only manifestation of that disease, but usually it appears as one of several manifestations. Rheumatic fever may produce all the varieties, viz., fibrinous, effusive, purulent, and chronic adhesive. Probably second in frequency as a causative factor is pneumonia, and in that disease it may be produced by metastasis or, in pleuro-pneumonia, by direct extension. With pleurisy affecting only one side it rarely occurs, but with double pleurisy it is an almost inevitable complication. It sometimes complicates empyema. Gonorrhea may be a more frequent cause than has been supposed. It occurs not infrequently with acute endocarditis or myocarditis by extension. It is occasionally found as a part of a local or general tuberculosis. It occasionally complicates scarlatina and the other exanthemata. Typhoid fever and erysipelas are among its rarer causes. It may occur secondarily to peritonitis and purulent conditions in the abdominal cavity, notably abscess of the liver. It may occur secondarily to inflammatory conditions in the mediastinum, to caries of the ribs or spine, and to phlegmonous inflammations in the neck; and pressure of an aortic aneurism may possibly produce it or predispose to it. It is not uncommon in chronic nephritis and gout. It may result from an external wound or from perforation by a needle, pin, bone, or other foreign body arriving by way of the esophagus. It occurs at all ages and is probably more common in men than in women.

Symptoms

The simple fibrinous variety may present no symptoms and be discovered accidentally, or there may be a slight pain in the precordium. After effusion has taken place the symptoms may be very slight, but usually they are moderate or severe. The symptoms of chronic adhesive pericarditis depend on the secondary hypertrophy and dilatation of the heart and the cardiac insufficiency.

The pain, which is not usually prominent in fibrinous pericarditis, may be severe when there is effusion. It may be a sharp and stabbing pain, or only a sense of distress and discomfort. Pressure over the precordium or the base of the sternum may aggravate it. It is often felt throughout the thorax, and is usually attended with a sense of oppression in the precordium. It is often referred to the epigastrium, the left shoulder, the left scapular region, or the left arm. Sometimes it occurs in paroxysms of an anginoid character, accompanied with great disturbance of the heart's action, dyspnea and coldness of the extremities.

Dyspnea may be a prominent symptom of pericardial effusion, and when it can not be accounted for otherwise, it suggests the diagnosis. It may be due to pressure of the fluid on the lung or to weakened heart action. With a large effusion the patient usually suffers less from dyspnea when in the sitting position, because of the gravitation of the fluid to the lower part of the sac and the consequent relief of pressure on the auricles and great vessels. In chronic pericardial adhesions the dyspnea is dependant on weakening of the heart muscle and interference with its action.

Symptoms other than dyspnea may be produced by large masses of fluid in the pericardium, viz., dysphagia from pressure on the esophagus, aphonia from pressure on the left recurrent laryngeal nerve, and irritable cough from pressure on the trachea.

In some cases nervous symptoms may be prominent. Restlessness and insomnia are common, and there may be delirium.

Fever may be present at the beginning and it may persist; it is always present when the effusion is purulent. Its course depends largely on the course of the causative disease. Occasionally there is hyperpyrexia.

Physical Signs

In simple fibrinous pericarditis inspection usually reveals nothing. Large effusions may produce fulness or bulging of the precordium, especially in children or in adults with thin chest walls. Subcutaneous edema over the lower end of the sternum is often seen. The impulse of the apex may be seen in the third or fourth interspace or it may not be seen at all. Very large effusions may depress the diaphragm and cause descent of the heart, in which case the apex beat may be below the normal and the left lobe of the liver may be pushed down so as to produce a prominence in the epigastrium (Anenbrugger's sign). The expansion of the left side of the chest is usually diminished. There may be pallor or cyanosis and the veins in the neck may be dilated.

Chronic pericardial adhesions may cause such enlargement of the heart as to produce precordial bulging and marked asymmetry of the chest. The extent of the cardiac impulse is increased, its character is usually wavy, and there may be systolic retraction of the chest wall in the region of the apex. Systolic retraction of the diaphragm when extensively attached to the pericardium, may be communicated to the chest wall and cause visible retraction of the latter (Broadbent's sign). This sign is usually most plainly visible in the tenth and eleventh interspaces posteriorly; less frequently in the seventh and eighth interspaces in front. Extensive adhesions to the diaphagm may also modify the normal respiratory movements in the epigastrium, and even cause them to disappear. Diastolic collapse of the jugulars is occasionally observed, but is not a distinctive sign of this condition.

A friction fremitus is often felt in acute fibrinous pericarditis, but sometimes it is not perceptible even when a friction sound is heard. When there is much effusion the most notable sign afforded by palpation is diminution or obliteration of the apical impulse. Friction fremitus disappears entirely after the appearance of the effusion, or it may persist at the base, or be felt only when the patient sits up. In chronic adhesive pericarditis a diastolic shock or rebound may be felt at the apex where a systolic retraction is visible.

With the effusion of fluid into the pericardium the area of cardiac dulness is observed gradually or rapidly to increase, sometimes to an extraordinary extent, the dull area being irregularly pear shaped, with the base directed downward, and the cardio-hepatic angle becomes obtuse (Rotchés sign). The lower lobe of the left lung may be so compressed by large effusions as to give rise to signs of consolidation when the patient sits upright, which disappear when the patient leans forward or lies on the right side. Similarly an area of dullness or skodaic resonance may be produced in the mid-axillary region. In chronic adhesive pericarditis the area of cardiac dulness is usually increased.

The most distinctively characteristic sign of fibrinous pericarditis is the friction sound, which is heard over the entire precordium or only in limited areas. It usually presents a quadruple rhythm with the two heart sounds, but it may be heard only with the systole. The effusion may cause it to disappear entirely or it may persist at the base or apex; and with absorption of the effusion it may reappear. The regular heart sounds are heard with diminishing distinctness as the effusion increases, but the pulmonary second sound is usually accentuated. Sometimes a systolic murmur is heard. Alteration in the breathing sounds due to compression of the lung is common.

The presence of inflammation in adjacent areas of the pleura may give rise to pleuro-pericardial friction sounds. When there is involvement of the mediastinum a creaking sound, heard over the sternum, may sometimes be elicited by up-and-down movements of the arms.

In chronic adhesive pericarditis there are no characteristic signs elicited by auscultation, but sometimes when the apex is adherent to the diaphragm and the base to the sternum, a peculiar presystolic tug, suggestive of a presystolic murmur, is heard over the apex.

The Course

Pericarditis often begins insidiously, but it may begin abruptly with pain or a chill, the latter being of regular occurrence when the inflammation is purulent. Simple fibrinous pericarditis rarely lasts longer than two weeks, and recovery apparently may be complete, or adhesions may remain.

The effusion may reach its height in two days and be rapidly absorbed, or it may slowly increase for several weeks. After absorption the peri- and epicardial layers may become adherent, or there may be adhesions to the sternum, diaphragm or pleura.

In severe cases which tend rapidly to a fatal termination, hyperpyrexia with intense dyspnea and cyanosis may develop. If the effusion becomes purulent, chills and a septic fever follow. In the acute fatal cases death usually takes place in a few days or a week, although it may be delayed for two or three weeks.

The course in chronic adhesive pericarditis depends on the changes which take place in the heart muscle. These are apt to be unimportant when the adhesions are limited to the peri- and epicardial layers, but with adhesions to the sternum, pleura or diaphragm, extensive hypertrophy and dilatation may develop, with cardiac insufficiency, and the course may be long and tedious.

Special Diagnostic Points

The to-and-fro friction sound is very nearly a pathognomonic sign in pericarditis, but not quite so, for there are a few other conditions in which it may be heard, viz., calcification of the coronary arteries and excessive dryness of the pericardial surfaces, especially in dilated and overacting hearts.

When the friction sound is heard only with the systole it may be mistaken for the murmur of mitral incompetence, but it can usually be distinguished from that murmur by its more strictly defined area of audibility and the absence of the other characteristic signs which go with that mitral lesion. Its superficial character is also a distinguishing point, as well as the fact that it synchronizes less exactly with the first heart sound.

From the double murmur of combined aortic stenosis and incompetence it is distinguished by its more strictly defined area of audibility, its less accurate synchronization with the regular heart sounds, and the absence of the changes in the area of cardiac dulness characteristic of the aortic lesion.

The pleuro-pericardial friction sound which is sometimes heard in pneumonia and also in tuberculosis, can easily be differentiated from the pericardial sign by the fact that it has a respiratory as well as a cardiac rhythm.

The area of dulness in pericardial effusion is differentiated from that in dilatation of the heart by its triangular shape, its extension upward along the left sternal border, and the fact that it shifts along its upper limits with changes of position. Particularly important in the differential diagnosis of effusion from dilatation, is the fact that in the former the heart sounds are distant and muffled, but not necessarily rapid or irregular, while in the latter the sounds are clearer, often more valvular in quality, and may be irregular: The circulation does not seem to be disturbed as much as would be expected with a dilatation producing increase in the area of cardiac dulness as great as that produced by a considerable effusion.

Pericarditis probably occurs much more frequently than it is diagnosticated: its frequently insidious onset, and the fact that its signs and symptoms are often overshadowed by those of the primary disease, make it easy to overlook it unless it is carefully watched for.

ACUTE ENDOCARDITIS

Acute inflammation of the endocardium is usually localized; it affects most commonly the free edges and the central faces of the valves, and the mitral more often than the aortic valve, and the endocardium of the left heart more often that that of the right. The inflammatory changes show extensive variations; the valves may be swollen and thickened; granulations may develop on them; the granulations may invade the mural endocardium adjacent to the valves; the granulations may become separated from their bases and be carried away by the blood stream as emboli; the endothelium in the inflamed areas may desquamate and be replaced by fibrin; ulcerations may develop in the inflamed areas; exhuberant proliferation may take place with production of friable and easily detachable emboli; the ulcerations may cause extensive erosion and even perforation of valves, of the interventricular septa, and of the cordae tendineae. The inflammatory process may extend so as to involve the myocardium and the pericardium.

A clinical distinction is generally made between the milder and severer types of acute endocarditis, the former being called simple endocarditis, and the latter, malignant, ulcerative or septic endocarditis.

Etiology

Acute endocarditis occurs most commonly as a complication of rheumatic fever. It is a comparatively frequent complication in scarlatina, pneumonia and erysipelas. It may occur in any of the exanthemata; also, in typhoid fever, malaria and gonorrhea; also in inflammations of the tonsils and throat; and in any condition where exists a purulent or septic focus. The cases due to rheumatic fever are apt to be of the milder type, and those due to infection with the pyogenic bacteria of the severer type.

Symptoms

The symptoms of acute endocarditis are, as a rule, insignificant; they depend chiefly on the amount of the valvular defect, the degree of myocardial weakness and the occurrence of embolism. Precordial pain, dyspnea and palpitation are sometimes present. Fever, other than that belonging to the causative condition, is not a prominent symptom, although in the absence of an active causative condition, slight or moderate fever may be suggestive.

Physical Signs

The physical signs are often slight and sometimes wanting; regularly so if there is no valvular involvement. Usually there is involvement of one or more valves. The murmurs characteristic of the valvular lesions have already been described, and will be summarized again in the description of the chronic valvular lesions; and so will be passed over here with the remark that, in general, the murmurs of acute endocarditis are less loud, less harsh and more blowing than those due to corresponding chronic valve lesions, and that they are apt to change from day to day with the changing pathological conditions.

Course

The course in simple endocarditis eventuates in apparently complete recovery in some cases, but very often permanent valvular defects are left. The course in malignant endocarditis may be short, or it may be prolonged for several months or a year; it is largely determined by the complications due to septic embolism.

Special Diagnostic Points

An important point in the diagnosis is to exclude functional conditions and conditions of acute myocardial degeneration which produce murmurs similar to those which develop in organic valve disease. Functional murmurs are softer than those due to organic valve disease, as a rule, and there is not usually found a coexisting enlargement of the heart. In cases of acute myocardial degeneration the diagnosis may be more difficult. In that condition there may be dilatation of the heart and accentuation of the pulmonary second sound with a mitral systolic murmur; but the dilatation is usually greater than in acute valvular endocarditis, and the murmur is usually softer and less liable to change its character, and the accentuation of the pulmonary second sound is not very marked. The pulse in the myocardial condition is apt to be small, of varying rapidity, and more or less irregular, and, does not respond readily to cardiac stimulants; while the pulse in acute endocarditis may show nothing abnormal except weakness and some acceleration.

The differential diagnosis of acute from chronic endocarditis is made by the history, and, in the acute condition, the presence of coexisting diseases, the changing character of the signs and the presence of fever.

The diagnosis of the severer types of the disease, the so-called malignant, ulcerative or septic endocarditis, often presents difficulties in the early stages. The murmurs may be very soft, and the fever may be slight, moderate, irregular, or, very rarely, absent. The course of the fever may resemble that of recurrent acute endocarditis, of typhoid fever, of meningitis, or of septicemia. Sooner or later, however, phenomena due to septic embolism reveal the diagnosis. In the prolonged cases of the malignant type there may be high fever, sweats and petechiæ, and small, painful nodules may appear under the skin. Important points in the diagnosis of malignant endocarditis are the pre-existence of a chronic valve lesion and the presence of a septic focus.

CHRONIC ENDOCARDITIS—VALVULAR DISEASE

Chronic endocarditis may develop as a primary condition, in which case the lesions consist of fibrosis or atheroma of the valves and adjacent structures, produced by nutritional changes or strain; or it may occur as a sequel of acute endocarditis. In both cases there is usually deformity of one or more of the valves. The valvular deformity is so regular a feature of this condition, and determines to such a large extent its symptoms and course, that the name valvular disease has been given to it by general usage.

The primary pathology in this condition consists of changes in one or more of the valves which impair their mechanical function, either by causing them to leak when closed, or to obstruct the flow of the heart stream when open; and the secondary pathology consists of changes in the size and shape of the heart chambers which the valvular defects produce by disturbances of the pressure relations inside the heart; changes in the myocardium which develop in response to altered mechanical, functional and nutritional conditions in the heart; and changes throughout the body due to venous stasis and ischemia.

Although the regular symptoms of cardiac insufficiency may develop in all the forms of valvular disease, there are some differences to be noted in them; and the physical sign groups show notable and characteristic differences. The following discussion of the special diagnosis of the individual valve lesions is very brief and cursory, because the subject has already been dealt with, although in its reverse aspect, in the discussion of endocardial murmurs.

MITRAL INCOMPETENCE

The characteristic physical signs are: Increase both to the left and right of the area of deep cardiac dulness; a systolic murmur, synchronous with the first sound, which is usually heard with maximum intensity at the apex, and is usually transmitted toward the axilla, sternum and base, and often to the back at the inner side of the left scapula near its tip, and may be widely transmitted over the precordium and chest; and accentuation of the pulmonary second sound, unless the right ventricle has failed.

The symptoms are chiefly due to passive congestion, and develop when compensation has failed or when the reserve power of the heart has been overtaxed. The lungs may be first congested, with production of a cough which is brought on by slight exertion. Later the lungs may be so congested as to cause dyspnea of a severe type. The liver may be congested and enlarged sometimes to an extraordinary extent and painful and tender. In the alimentary canal and kidneys passive congestion may produce functional disturbances and even structural changes. The lower extremities may become edematous, and, as decompensation progresses, the edema may extend upward and become general and extreme. Passive congestion in the brain may cause orthopnea and also nervous and mental symptoms. Subjective symptoms referred to the heart are rare except in neurotic subjects. As a rule, decompensation develops more slowly in mitral incompetence than in the other valve lesions, and the patient has more warning of impending disaster; and he can, as a rule, stand a greater degree of decompensation and more frequent recurrences of it and recover.

The differential diagnosis from dilatation of the heart due to myocardial degeneration or weakness, and from functional conditions producing a systolic murmur, may be difficult if the organic mitral lesion is slight in extent. From the former it is made chiefly by the history, the harsher and more persistent character of the murmur in organic valve disease and its greater area of audibility, and the more pronounced accentuation of the pulmonary second sound; and from the latter, by the absence of the characteristic alterations in the area of cardiac dulness that belong to the organic valve disease, in addition to the points just mentioned.

MITRAL STENOSIS

Stenosis of the mitral valve rarely occurs without a certain amount of incompetence.

The characteristic physical signs are: Increase to the right of the area of deep cardiac dulness; a presystolic thrill; a systolic shock at the apex; a short, snapping first sound at the apex; accentuation of the pulmonary second sound; and a diastolic-presystolic, or mid-diastolic-presystolic murmur heard with maximum intensity a little inside the apex, and usually not far transmitted, though exceptionally it may be widely transmitted. This murmur frequently is not heard, and may appear and disappear, and is, in fact, the most inconstant of organic heart murmurs. A reduplication of the second sound is often heard only at the apex. With progressive loss of compensation, there may be extension of the area of deep cardiac dulness to the left. The pulse, which is regularly small, shows early a tendency to irregularity, and in the last stages becomes absolutely irregular, in consequence of the development of auricular fibrillation.

The symptoms due to passive congestion differ somewhat from those in mitral incompetence, particularly in the increased tendency to the early development of dyspnea. Breathlessness or a sharp pain in the region of the apex on unusual exertion are common and early symptoms. There is a tendency to attacks of bronchitis and palpitation. After compensation has been lost the symptoms are practically the same as in mitral incompetence, except that edema of the lower extremities is less pronounced and general anasarca less frequently observed; although enlargement of the liver occurs regularly, and often to an extreme degree, with ascites.

The course, in general, is shorter than in mitral incompetence, and more likely to be interrupted by accidents, including embolism. The writer, in all his experience, has seen but two patients with mitral stenosis who had passed their fiftieth year. Loss of compensation is produced more easily, and is recovered from with more difficulty, than in mitral stenosis.

The differential diagnosis from mitral incompetence is not usually difficult, except in the stage of lost compensation. Then the fact that the secondary results of the valvular defect are apt to be more severe than in incompetence may have significance. In the absence of the characteristic murmur or thrill, the diagnosis can often be made on the history, characteristic alteration of the cardiac outline, and the snapping quality of the first sound at the apex.

AORTIC INCOMPETENCE

The characteristic physical signs are: Enlargement of the left ventricle downward and to the left, which is usually extensive, and may be extreme; an enlarged and forcible or heaving apex beat; visible pulsation of the carotid and other arteries, and of the capillaries under favorable circumstances; a full, bounding pulse, which collapses quickly under the finger; a diastolic murmur, which is heard with maximum intensity in the aortic area, in the third or fourth left interspace at the sternal edge, at the base of the sternum, or at the apex, and which is transmitted, slightly or extensively, down the sternum, toward the apex, and upward over the course of the great arteries. There is often heard in this condition a systolic murmur, with point of maximum intensity in the aortic region and transmitted upward, which does not necessarily mean stenosis, but roughening of the aortic valves; and sometimes a presystolic murmur, first described by Flint, which does not necessarily mean an organic mitral stenosis.

The symptoms, while compensation remains good, are chiefly due to the hypertrophied heart, which is easily made to overact and produce throbbing in the neck and head, headache, vertigo and ringing in the ears; and to the ischemia: the skin is usually pale, in contradistinction to the cyanosis, which often appears in patients with mitral lesions. Palpitation and a sense of oppression, and, especially in the arteriosclerotic or degenerative cases, pain in the precordial region which may radiate to the shoulders and arms, are not uncommon symptoms. After compensation has been lost there is the usual train of symptoms due to pulmonary and venous stasis, but edema of the lower extremities is not much in evidence, and when present rarely extends beyond the feet and legs. Symptomatic anemia is usually present in a comparatively high degree, and may be responsible for the slight edema in some cases. Peevishness, irritability and melancholia are common.

The course varies greatly. In cases following acute endocarditis in youth the course may be long, and the patient may show an extraordinary capacity for severe physical exertion; but sudden death occurs more often in this than in any other of the valve lesions. In the degenerative cases which develop late in life, especially those in which there is sclerosis of the coronary arteries, the course may be short.

AORTIC STENOSIS

The characteristic physical signs are: Enlargement of the area of cardiac dulness to the left and downward; a systolic thrill felt with maximum intensity in the aortic area, but often perceptible over the entire precordium and the carotic and subclavian arteries; a loud, harsh, late systolic murmur, heard with maximum intensity most often in the aortic area, but sometimes over the apex, which is transmitted upward over the course of the carotids; a faint or inaudible second sound; and a small, rather slow pulse. Incompetence of the aortic valve is often associated with stenosis.

The symptoms appear after failure of compensation and resemble more or less those of aortic incompetence. They may be delayed for many years.

The differential diagnosis is chiefly from a roughened condition of the aorta or the aortic valves, or a dilatated aorta, and from chronic nephritis with arteriosclerosis and hypertrophy of the left ventricle; and is usually determined by the character of the murmur, which is less harsh in the other conditions than in the stenosis, and the character of the aortic second sound, which is usually distinct or accentuated in those conditions.

TRICUSPID INCOMPETENCE

Relative incompetence of the tricuspid valve commonly develops in the course of the breakdown of the heart from any of the left heart valve lesions; but as a primary condition it is extremely rare. The characteristic physical signs are: Pulsation of the jugular veins, which is systolic in time; expansile pulsation of the liver, extension of cardiac dulness to the right; and a soft, systolic murmur, heard with maximum intensity at the lower end of the sternum, which is transmitted a short distance to the left and a short distance upward. The second pulmonary sound is usually faint. Cyanosis is common in this, as in all right heart valve lesions.

TRICUSPID STENOSIS

The characteristic physical sign of this, the rarest of the valve lesions is a presystolic murmur or rumble which is heard within a limited area at the lower end of the sternum. It is usually associated with other valve lesions whose physical signs predominate.

PULMONARY INCOMPETENCE

The physical signs of this rare lesion are: Extension of the area of cardiac dulness to the right, and a diastolic murmur which is most audible in the second left interspace near the sternum, and which is transmitted down the sternum. This murmur can with difficulty be distinguished from the murmur of aortic incompetence, and the differential diagnosis is made by the absence of the water hammer pulse and the absence of hypertrophy and dilatation of the left ventricle.

PULMONARY STENOSIS

This condition, though rare, is the most frequently observed of all the primary congenital heart valve lesions. The characteristic physical signs are: Moderate extension to the right of the area of cardiac dulness and a harsh, systolic murmur which is best heard with maximum intensity in the pulmonary area, and which is transmitted a short distance to the left and upward. It is also frequently heard in the back between the scapulae, and may be widely audible. Usually a systolic thrill is felt in the pulmonary area.

COMBINED VALVE LESIONS

Most valvular lesions, if not originally combined, become so before their course is finished. Incompetence usually develops in the stenoses. Relative incompetence develops in the mitral valve as decompensation in aortic lesions progresses, and in the tricuspid valve as a sequel to incompetence or stenosis of the mitral or pulmonary valve. Sometimes co-existing valvular lesions compensate for each other to a certain extent; mitral stenosis and mitral incompetence, and aortic stenosis and aortic incompetence may do this; and relative mitral incompetence may temporarily relieve over distension of the ventricle in aortic incompetence. But some combinations, like mitral stenosis and aortic stenosis, or mitral incompetence and aortic stenosis, intensify the disability. Relative tricuspid incompetence is usually a serious complication in lesions of the left heart with lost compensation.

The characteristic physical signs of combined valve lesions are those of the individual lesions, more or less confused. Usually the signs of the chief lesion can be made out.

CONGENITAL DISEASES OF THE HEART

Congenital valve diseases of the heart, which are the result of fetal endocarditis, usually affect the valves of the right heart; these lesions have already been discussed. Other congenital lesions are of the nature of arrests of development, deformities or displacements. Of these the most important are: Absence of septum between the two auricles or the two ventricles; patent foramen ovale, which is often associated with pulmonary stenosis, patent ductus arteriosus; and abnormal semilunar valves, the cusps of which may be only two in number, or more than three.

The most constant and striking sign of congenital heart disease is cyanosis. This sign has given its name to the "blue baby." The fingers may be clubbed. The murmur, if produced by obstruction at the pulmonary orifice, is apt to be loud and harsh and heard with maximum intensity in the pulmonary area; while if produced by a defect of the septum, which does not always give a murmur, it is usually loud, systolic in time, and is regularly heard over the entire precordium. The area of cardiac dulness may not be much increased.

The chief diagnostic points are: The prevalence of cyanosis with comparatively little dyspnea; the loud character of the murmur, if present; comparatively little dilatation, as a rule; and the history of persistence of the condition since birth.

ACUTE MYOCARDITIS—ACUTE MYOCARDIAL DEGENERATION

The myocardium may be the seat of acute degenerative changes of various kinds from cloudy swelling to the formation of abscesses. The morbid processes are apt to be localized, and occur with particular frequency in the papillary muscles of the left ventricle and the musculature of the auriculo-ventricular ring. When abscesses form they are usually due to septic thrombi which plug small arteries. Embolism of a coronary artery may produce coagulation necrosis of a considerable portion of the myocardium.

Etiology

Acute myocarditis is observed most frequently as a complication of the acute infectious diseases, notably, diphtheria, typhoid fever, influenza, pneumonia and scarlet fever, and particularly rheumatic fever, in which it may be also produced by extension of an acute endocarditis or pericarditis. Pyemia and conditions in which there is a septic focus are among its causes.

Symptoms

The symptoms are usually insignificant in comparison with the gravity of the lesions. They may be so slight as to escape notice.

Among the symptoms which are sometimes observed are: Restlessness, apathy, palpitation, syncope, precordial pain, vomiting and the regular symptoms of weakened heart power.

Physical Signs

The skin may be pale; the action of the heart may be tumultuous, especially in the early stages; there may be dilatation, which, taken in connection with the tumultuous action, is suggestive of the diagnosis; the apex beat may be feeble or absent; the heart sounds may be short, sharp, feeble, muffled or embryo cardial; murmurs are sometimes present, which do not seem to depend always on dilatation, but on incoordination of different parts of the heart muscle; and the pulse is generally rapid, though sometimes it is slow, and it is usually small, compressible and irregular. Unusual exertion, even movements of the bowels, may have an exaggeratedly depressing effect on the action of the heart.

Course

Sudden death may occur at any time in the course. Apparent recovery may take place in the milder cases, especially those in which the lesions are circumscribed, after variable periods; or a condition of chronic myocardial degeneration may persist. The period of convalescence in a favorable case, which has shown definite signs and symptoms, may be a matter of months.

Special Diagnostic Points

It is often impossible to make the diagnosis. When the signs and symptoms which have been mentioned develop suddenly in the course of an acute infectious disease, the condition may be suspected. If they develop during convalescence from those diseases they possess even greater diagnostic significance.

CHRONIC MYOCARDITIS—CHRONIC MYOCARDIAL DEGENERATION

Chronic myocarditis, including in that term all forms of chronic myocardial degeneration, dominates the pathology of the latter part of life, as valvular disease does that of the first part.

The characteristic pathology consists of fatty or fibrous degeneration of muscle fibres; invasion of the fibre bundles by strands of connective tissue, thickening of connective tissue septa between the muscle strands; and cicatricial formations in isolated patches of inflammation which have healed and in areas of infarction which have organized. Fibrosis may follow fatty or parenchymatous degenerations which have destroyed muscle tissue; and it may encroach on the muscular element in the reparative stage of an acute myocarditis; and it may be a conservative process. The characteristic changes may be diffused throughout the heart, but are usually confined to limited areas, particularly in the wall of the left ventricle, the papillary muscles and the interventricular septum. Circumscribed thinning and dilatation of the heart wall, and the formation of cardiac aneurisms, may result from fibroid degeneration. There is often associated sclerosis of the coronary arteries, with a tendency to the production of thrombosis and anemic infarcts. Hypertrophy and dilatation are both liable to occur, and organic valvular lesions may result from sclerosis, and functional ones from dilatation and muscular incoordination. Sclerotic changes may cause contraction of the conus arteriosus and narrowing of the aortic and pulmonary orifices.

Etiology

Chronic myocarditis may follow the acute condition, but more often it develops independently. Its etiological factors are for the most part the same as those of arteriosclerosis; and coronary sclerosis enters into the immediate causation in a large proportion of the cases. Lesions of the coronary arteries or their branches may diminish the blood supply of particular regions of the myocardium, thereby diminishing their nutrition and inviting fatty or fibroid degeneration; or, by thrombotic occlusion, may completely deprive the regions affected of their blood supply, transforming them into areas of myomalacia cordis. Interference with the coronary circulation also may be produced by valvular disease. Some poisons may act directly on the myocardium to produce chronic degenerative changes, among which are: Alcohol and tobacco; the poisons present in the blood as a result of gout, diabetes, chronic nephritis, and relatively excessive putrefaction in the alimentary canal; the specific poison of syphilis, and toxins from a focus of chronic bacterial infection. Chronic myocarditis, also, may be caused by direct extension of the infective inflammatory process in chronic endocarditis and pericarditis. Acute and chronic cardiac overstrain, and hypertrophy and dilatation from any cause, predispose to it, as do also anemia and malnutrition.

Symptoms

Usually the first symptom noted is shortness of breath on moderate exertion, such as going upstairs or climbing a hill, or breasting a cold wind after eating a hearty meal. The dyspnea may increase with progressive increase of the myocardial weakness and there may be attacks of cardiac asthma; and the regular symptoms of general venous congestion may develop. Palpitation, a feeling of heaviness or constriction in the precordium, and a sense of fatigue after slight exertion, may be among the early symptoms. Mental inertia is common, and in the later stages of the disease there may be mental disturbances and even chronic mania. Attacks of angina pectoris of all grades of severity may occur. There may be no symptoms until sudden death reveals the condition.

Physical Signs

There is usually some increase in the area of deep cardiac dulness. The apex beat is apt to be feeble, or to present the characteristic signs of hypertrophy or dilatation. The heart sounds are apt to be soft and impure; or, if there is hypertrophy, the first sound at the apex may be prolonged and of low pitch, and the second sound clear and ringing; while if dilatation predominates, the first sound at the apex is short and sharp, closely resembling the second and the effect of embryocardia is produced. With increased feebleness of heart action the sounds may become faint muffled. Occasionally the gallop rhythm is heard in the neighborhood of the left nipple, especially in cases complicating chronic nephritis. Murmurs may appear, which are due to atheromatous roughening or relative incompetence. The pulse is often regular, but irregularities are frequently observed in it. There may be bradycardia, sometimes combined with intermittency, or the signs of heart block. Extra systoles, more frequently ventricular than auricular, may be present. The pulse may be persistently accellerated, or tacchycardia may occur in paroxysms. There may be an absolute arrythmia. The blood pressure may be normal, but usually it shows a tendency to be lower than is required by existing conditions, with an unduly large pulse pressure.

Course

The patient may live for many years after definite signs have appeared, but is liable to have his course cut short by rapidly progressive development of cardiac insufficiency, by an attack of angina pectoris, by coronary thrombosis, by acute dilatation, by rupture of a cardiac aneurism, or by some intercurrent disease which he could not successfully resist on account of his myocardial weakness. The carefulness with which he regulates his life has much to do with the course.

Special Diagnostic Points

If the characteristic symptoms and signs are abundantly present, the diagnosis may not be difficult, but usually they are not abundantly present, and may be altogether absent; and very often the diagnosis, if made at all, can be only a probable one, based on a study of scanty evidence, some of which may be equivocal.

Age is of special importance in the diagnosis. Cardiac insufficiency developing in a person past middle life, in whom no evidences of endocarditis or pericarditis, past or present, can be found, suggests chronic myocarditis, and signs of cardiac insufficiency developing in a person past middle life, in whom are found evidences of chronic nephritis or arteriosclerosis, are strongly suggestive of myocardial disease.

The diagnosis, in any case, may be rendered more probable by the history, if it includes syphilis, alcoholism, prolonged cardiac strain or other recognized etiological factors.

The comparative frequency with which enlargement of the outline of deep cardiac dulness assumes a more or less quadrangular shape in chronic myocarditis, suggests an aid to the diagnosis; and the softening of the heart sounds, which is often observed, and particularly the weakening and shortening of the first sound at the apex, so as to produce the effect of embryocardia, are among the more significant signs. Arrythmias observed in elderly persons, especially if associated with other signs of cardiac disease, are suggestive of chronic myocarditis.

HYPERTROPHY OF THE HEART

In hypertrophy of the heart there is increase in size of the muscle fibres without increase in their number; with a certain amount of degeneration in some of them; and with a certain amount of proliferation of the connective tissue between the fibres. As met with clinically, it is almost always secondary to some other condition which calls for continued extra work on the part of the heart: rarely it seems to be produced in a healthy person by hard physical work, as in professional athletes. It is regularly a conservative process.

There are no symptoms directly due to the hypertrophy and when symptoms develop, it is usually an indication that compensation is beginning to fail.

The characteristic physical signs are: Displacement of the apex to the left, or to the left and downward, according to the heart chambers involved and the extent of the hypertrophy; a strong, rather broad apex beat; a loud, booming, rather prolonged first sound, and accentuation of the second sound; and, when the hypertrophy is of the left ventricle, a full, often rather slow, high tension pulse. With hypertrophy of the right ventricle there is usually marked epigastric pulsation.

DILATATION OF THE HEART

Dilatation of the heart means increase in the capacity of its chambers and is due to loss of tonicity of the heart muscle. It develops regularly after loss of cardiac compensation, and occurs as a terminal condition in most cases of heart disease. It may be compensatory and accompanied by a compensatory hypertrophy, as in aortic incompetence, and in athletes who develop a capacity for feats of endurance. It may occur transitorily, as after an unusual muscular effort; and it may be produced acutely by a severe or continued muscular overstrain, and not be promptly recovered from, but remain as a long continued or permanent disability.

The symptoms are those of cardiac insufficiency and venous congestion; and, especially in the acute cases, there may be dyspnea, a feeling of exhaustion, palpitation, precordial discomfort or pain, and perhaps an unusual tendency to yawning.

The characteristic physical signs are: Increase in the area of deep cardiac dulness; a feeble, diffuse and wavy apex beat; rapid and feeble heart sounds, which may be irregular and intermittent, or embryocardial in type; diminution in intensity of the aortic second sound; and, perhaps, murmurs due to relative incompetence of the auriculo-ventricular valves.

FATTY HEART

The term fatty heart is applied by general usage to two pathological conditions. In one, there is an extensive increase in the amount of the sub-pericardial fat and of that between the muscle fibres of the heart, which occurs as part of a general obesity. In the other fat globules appear inside the muscle cells, and are either due to fatty degeneration of the muscle cells (in which case the condition belongs nosologically with the myocardial degenerations previously discussed), or to a disturbance of cellular metabolism, which prevents the proper combustion of fat globules which have found their way into the muscle cells from without.

The etiology is that of obesity (which is a perversion of fat metabolism), and perhaps, also, especially in the cases characterized by intracellular deposits of fat, that of chronic myocardial degeneration, particularly as regards chronic alcoholism, excessive beer drinking, luxurious living, a sedentary life, and anemia.

The signs and symptoms are not sufficiently distinctive to be of much aid in the diagnosis, which, however, can usually be made by inference from the etiological factors. There is apt to be general debility and some shortness of breath on exertion. The heart may or may not be dilatated. The sounds at the apex are feeble and distant or embryocardial in character. The pulse is small and feeble and the blood pressure low unless elevated by some complication.

ANGINA PECTORIS

There are certain symptoms or syndromes involving the heart which stand out so clearly and with such prominence, that they have been conceded by general usage a name and place among the cardiac diseases. Such are angina pectoris, Stokes-Adams disease, paroxysmal tachycardia, and exophthalmic goiter.

The characteristic syndrome of angina pectoris consists of a sudden attack of intense pain in the region of the heart, with a feeling as if a tight band were drawn around the chest, radiation of the pain to the neck and arms, and a sense of impending death. These symptoms appear in varying degrees of severity and in the severer degrees they are probably the most agonizing that humanity is called on to bear.

In most of the cases which are fatal in a first or a recurrent attack, the pathological basis is coronary sclerosis. Among the less common organic causes are aneurism, especially of the ascending aorta; valvular disease, especially aortic incompetence and mitral stenosis; and adherent pericardium. In a group of cases which seem to be due to arterial spasm without coronary sclerosis or other demonstrable organic lesion, the prognosis may be good. The cases of this group may occur as a result of disturbances of the gastrointestinal tract, of hysteria, neurasthenia, and the abuse of tobacco, coffee and tea. Perhaps to this group belong the cases which are sometimes observed in hyperthyroidism. In acute cardiac overstrain with dilatation attacks of angina pectoris may occur, which seem to possess no essentially malign significance.

The diagnosis of the syndrome itself is usually easy; but the differential diagnosis between angina due to coronary sclerosis or other organic heart lesion, and that due entirely to functional disturbance, may present great difficulties. Even in the absence of demonstrable evidence of organic disease, if the patient is past middle life, it is safest always to suspect an organic element in the etiology.

STOKES-ADAMS DISEASE

The syndrome called Stokes-Adams disease has for its essential features, a very slow pulse with attacks of syncope or convulsions. It is produced by auriculo-ventricular heart block, with temporary cessation of ventricular contractions, or unusual protraction of an interval between the contractions. The heart block is responsible for the slow pulse, and the unusually protracted periods of asystole, for the cerebral anemia and syncope. The attacks characterized by this syndrome may occur at the transition from normal rhythm to complete block, in the midst of a complete block,and it is probable, also, from stimulation of the vagus in certain cases where conductivity is already diminished.

The diagnosis is made by finding an extremely slow radial pulse in a patient who has a sudden attack of syncope or convulsions. The pulse rate may be as slow as thirty, or twenty, per minute, or even slower. The diagnosis is suported by a history of syphilis, or evidences of arteriosclerosis or chronic myocarditis.

PAROXYSMAL TACHYCARDIA

The characteristic features of paroxysmal tachycardia are: The increased pulse rate, which is usually twice, thrice or four times the customary rate; the suddenness with which the increase develops and ceases; and the persistently regular character of the rapid pulse. The pathological basis of this syndrome seems to involve increased irritability of the cardiac muscle, reflex stimulation of cardiac nerves, and sometimes auricular fibrillation. The contractions may be of the nature of extra systoles. The symptoms are not usually pronounced, but there may be palpitation and a feeling of oppression in the precordium; or dyspnea, which may be present without increase in the respiration rate; or weakness, restlessness, vertigo, and sometimes syncope; or, if the attack is of long duration, symptoms of venous congestion. The face is usually pale. The area of cardiac dulness is not usually increased, though in some cases it may be so near the end of the attack. The heart sounds are embryocardial in character, and a soft, systolic murmur is sometimes heard in the mitral or tricuspid area. The duration of an attack varies from a few minutes to a few days. The attacks occur at all ages, and are usually found unassociated with demonstrable heart lesions.

The diagnosis is usually easy except in the cases with pulse rates of only about 140 or 150: to make the diagnosis in such cases it is necessary to observe the onset and termination of the tacchycardia: a sudden rise to the maximum pulse rate in a few seconds, and as rapid a return to the former rate, suggest the syndrome.

EXOPHTHALMIC GOITER

The striking syndrome of tachycardia, bulging eyeballs and enlarged thyroid gland, generally known as exophthalmic goiter, Basedow's disease, or Graves' disease, is a manifestation of hyperthyroidism. It is included among heart diseases by general usage, because of the prominent place in the syndrome occupied by the cardiac symptoms. Usually found associated with the three cardinal symptoms are tremor and nervous excitability. The diagnosis rarely presents any difficulties.

CARDIAC NEUROSES—FUNCTIONAL DISORDERS OF THE HEART

Certain disorders of the heart which are apparently due to disturbing impulses which come to it through its nervous connections, and for which no basis in organic heart changes can be found, are classed, pending increase in pathological knowledge, as cardiac neuroses. The disturbing impulses may come from anatomical derangements which interfere with the free action of the heart, viz., flat chest, curvature of the spine, pleuritic adhesions, obesity, tight lacing, abdominal flatus, and an unusually long thorax or a depressed diaphragm (due to enteroptosis) which produces cardioptosis. They may come as a result of visceral reflexes, viz., from swallowed air, which also may act mechanically to raise the diaphragm, and from irritation of the stomach and intestines by their contents, particularly by excess of hydrochloric, lactic and butyric acid.. They may come as reflexes from the sexual organs. They often come from toxemic irritations, particularly those produced by the intestinal toxins, and by alcohol, coffee and tobacco: the writer has seen anginoid symptoms brought on in a neurotic patient by a few whiffs of a cigar. They may be due to anemia. They occur frequently as a result of emotional excitement.

The symptoms are sensory and motor. The former are the most common, or, at least, the ones most often complained of by the patient. They include: Palpitation, tenderness over the precordium, abnormal sensations in the region of the heart, particularly pain, a sense of constriction and symptoms simulating those of angina pectoris, and sometimes sensations of a strange and fanciful character. The principal symptoms due to motor disturbances are those associated with cardiac arrhythmia and weakness of the heart action, including among the latter, fainting spells. Neurasthenic and psychasthenic symptoms are often associated with the local cardiac symptoms. The blood pressure is usually low.

The diagnosis, which is not always easy, is made chiefly on the symptoms, the presence of a source of nervous irritation, and the absence of signs of an organic heart lesion. If evidences of a heart lesion are found, it is well to give them full credit, even though they may be apparently overwhelmed by the neurotic symptoms. A mitral systolic murmur, however, which is audible only when the heart is overacting, and which is unaccompanied by a permanent increase in the area of cardiac dulness, usually may be interpreted as of functional origin; and cardiac arrythmia, of which the patient is subjectively conscious, in the absence of other signs of organic heart disease, can usually be referred to a functional origin; but, if the arrythmia is not attended with subjective sensations it is safest to interpret it as an evidence of myocardial disease. Ability to undergo physical exertion of considerable severity without dyspnea, suggests a functional condition. The neurotic character of a cardiac pain may be suspected if it originates spontaneously, that is, independently of physical exertion or mental excitement. A personal or family history of neurasthenia, hysteria, hypochondria or the neurotic temperament, strengthens the diagnosis of a functional disorder. In any case where the patient is past middle life the diagnosis must be doubtful.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOBLISM

Antitrypsin Reaction in Clinical Diagnosis—G. WEIL, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

The result of 151 cases in which the antitrypsin reaction was performed. The reaction is not specific for any pathological condition, but it has a certain significance in pregnancy, in gastric affections and suppurative processes. MILL.

Test for the Demonstration of a Large Increase of Leukocytes in the Blood—A. BITTORF, Deutsche med. Wochenschr., Aug. 31, 1916.

Decinormal sodium hydrate solution is placed into a Sahli hemoglobin tube to the mark 10-30. To this is added by means of the little suction tube coming with the apparatus 20 to 40 cubic millimeter blood. This is moderately shaken. In case the leukocytes are greatly augmented, the mixture becomes of a gelatinous consistency as the result of the swelling of the leukocytes. MILL.

Increased Blood Pressure and Physical Blood Changes—K. KLEEBERGER, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVIII, No. 2.

Observations on 8 cases. Blood pressure increase may be entirely independent of an increase in molecular concentration of the blood. Molecular concentration is dependent on the nephritic state. The osmotic control of the tissues is not exercised by the arterial membranes, but by the capillaries. The viscosity may show fluctuations which are not due to molecular concentration nor to blood pressure increase. Blood pressure increase cannot be explained on the hand of physical alterations of the blood. WESTERN.

Physiological Activity of Thyroid Secretion—L. ASHER, Deutsche med. Wochenschr., Aug. 24, 1916.

The vaso-constricting activity of the blood of Basedow patients does not depend upon an increase of adrenalin, but upon the activation of the efficiency of the adrenalin already present. This activation is caused by the increased content of thyroid secretion. MILL.

Effect of Iodthyroglobulin and Thyronucleid on the Nitrogen Metabolism and the Blood-Picture of Myxedema and Basedow's Disease—H. COURVOISIER, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

In a case of myxedema there was found after administration of thyronucleid, retention of nitrogen and aggravation of the blood-picture. When iodthyroglobulin was administered the nitrogen excretion became increased and the blood-picture improved. After administration of a mixture of both substances there ensued a successive effect. In two cases of Basedow's disease iodthyroglobulin caused augmentation of nitrogen metabolism and an aggravation of the blood-picture, while the administration of thyronucleid was followed by a diminished nitrogen metabolism and an aggravation of the blood-picture. MILL.

The Effect of Some Thyroid Preparations on the Metabolism and the Blood-Picture of Myxedema and Cretinism—W. LANZ, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

Of all the preparations of the thyroid gland iodthyroglobulin and the preparations containing it are the only ones which exert an influence upon metabolism. Iodin per se shows no metabolic influence. MILL.

The Effect of Thyroid Preparations on Thyrodopaths—A. KOCHER, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

The iodin-containing preparation of the thyroid is the only one which influences all the pathologic symptoms of the thyrodopath. The toxic symptoms following the administration of the entire substance are much less marked. There are other active substances in the thyroid which influence the blood or favorably influence the thyroid proteid. MILL.

The Blood-Picture in Goiter and its Alteration after Operation—M. JASTRAM, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

Examination of 58 cases of goiter showed the presence of lymphocytosis in every instance. This lymphocytosis runs parallel with the severity of the symptoms. Generally speaking, the operation of simple goiter is not followed by a decline of the lymphocytosis. In Basedow's disease, on the other hand, the operation causes a diminution of the lymphocytosis. MILL.

Addison's Syndrome in Old People—A. ARAVANTINOS, Deutsche med. Wochenschr., Aug. 17, 1916.

The characteristic symptoms of Addison's disease are often met with in old age. The pigmentations in old age, mostly patchy or rather diffuse, indicate beginning functional deficiency of the suprarenal glands. MILL.

Diabetes Innocuus—G. ROSENFELD, Berliner klin. Wochenschr., Oct. 2, 1916.

A number of points seem to indicate that diabetes innocuus is of suprarenal origin. The pertaining cases are, of course, rather mild in character and should be strictly separated from those of pancreatic diabetes. Prognostically they are favorable. MILL.

Diabetes and War—A. ALBU, Deutsche med. Wochenschr., Aug. 17, 1916.

There exists no typical war diabetes. The special war injuries and conditions may, however, give rise to diabetes in those who are disposed to it. MILL.

Mortality from Diabetes Mellitus—H. MORRISON, Bost. Med. and Surg. Jour., July 13, 1916.

In a statistical study of the mortality from diabetes mellitus in Boston from 1895 to 1913, the ratio of the number of deaths due to this disease to the total number of deaths from all causes is 0.018 among Jews and 0.007 among non-Jews. The largest number of deaths from this disease was in those of Irish parentage. They numbered more than one-third of the total deaths. There has been a steady rise in the death rate from diabetes mellitus in Boston. SACHS.

Acetone Body Production in Children—J. HOWLAND and W. McK. MARRIOTT, Am. Jour. Dis. Child., Nov., 1916.

Acidosis and acetonuria are not synonymous terms. There is no justification for believing that acidosis is due to acetone bodies occuring in epidemic form. Deficient food or increased requirement for food (disproportion between caloric intake and output) is the chief cause of acetonuria, but this rarely results in acidosis. The production of acetone occurs at times when starvation cannot be held responsible. The production is rapid and excessive and sufficient to cause a severe or fatal acidosis. This condition is not necessarily accompanied by vomiting. It probably depends on the same underlying metabolic disturbance as do the majority of cases of recurrent vomiting. SACHS.

Lipodystrophia Progressiva—J. GERSTMANN, Wiener klin. Wochenschr., Sept. 21, 1916.

Description of a very rare affection which occurred—exceptionally—in a man. The condition started in early youth and progressed very slowly from the head to the trunk. The lower extremities did not become involved; on the contrary, marked fat deposits were found in this region. The thyroid gland was found not to functionate. The musculature of the diseased parts of the body was exceedingly well developed. In certain regions the muscular development was even excessive. MILL.

Familial Hydrops Intermittens and Purin Metabolism—H. C. FRENKEL-TISSOT, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVIII, No. 1.

Two sisters showed manifestations of intermittent hydrops genus. A third sister showed no pathologic symptoms at the same period, but a fourth sister suffered from periodic attacks of hemicrania which were probably an equivalent of the arthritic swellings of the sister. This seems to be a familial form of hydrops genus intermittens, a description of which is not to be found in literature. That the condition is of a vasoneurotic origin is shown by the severe accessoriustic, which was present in one of the sisters at the same time, as well as by the attacks of hemicrania and the occasional urticaria in one of the sisters as well as by the periarthritis destrues, prevailing in the mother. Metabolic examinations in two of the patients demonstrated disturbances of the uric acid excretion. These disturbances resembled those which are generally found in genuine gout, in various nervous and exanthematous affections.

WESTERN.

Digestion-Lipemia—J. COHN and W. HEIMANN, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVIII, No. 2.

Fat in the serum is much increased after ingestion of butter and cream. The cholesterin and lecithin contents of the serum (lipods) is not increased during the digestion-lipemia (lipods). The lipemic serum is due to the augmentation of the fat on the surface and not to the cholesterin. WESTERN.

Increase of the Effect of Narcotics by Cannabis Indica—A. GISEL, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVIII, No. 1.

The experiments were made with tincture of cannabis indica. The effect due to the alcohol contained in the tincture was duly considered. Seven c.c. of the tincture of cannabis indica alone frequently produced paralytic phenomena of a light or severe degree. When 8 c.c. of the tincture were injected, the experiment animals became often very restless and had apparent visions. This exalted state was not followed by tranquil rest. Very small quantities of urethan which, per se, exert no narcotic influence, give rise to a marked narcosis if administered with 6 c.c. tincture of cannabis indica. The effect of cannabis becomes already increased by the alcohol contained in the tincture. Combined administration of cannabis with morphin hydrochlorate is followed by a much increased narcotic total effect. WESTERN.

Determination of Nitrogen in Urine—R. B. H. GRADWOHL and A. J. BLAIVAS, Jour. A. M. A., Sept. 9, 1916.

This method is a slight modification of the method given by Meyers and Fine, which in turn is a modification of the calorimetric method of Folin and Farmer. The only difference in technic is the addition of hydrogen peroxid to hasten oxidation which shortens the time of completing the determination from 15 to 20 minutes to 5 to 10 minutes. The method is given in full in this article with a table for the estimation of the nitrogen with the Hellige colorimeter. SACHS.

Influence of Age and Sex on Hemoglobin—C. S. WILLIAMSON, Arch. Int. Med., Oct., 1916.

The amount of hemoglobin in the blood varies according to the age. The greatest variations are observed from birth to 16 years of age. There is a marked difference between the two sexes in this regard between 16 and 60 years, but this difference grows less after the sixtieth year. Thus it is evident that hemoglobinometers must be standardized in absolute terms, such as in grams of hemoglobin per 100 c.c. of blood. SACHS.

Functions of Cerebrospinal Fluid—W. D. HALLIBURTON, Lancet, Nov. 4, 1916.

The cerebrospinal fluid is normally formed by the secreting cells which are so prominently found covering the choroid plexuses in the cerebral ventricles. The true cerebrospinal pressure is the result of the secretory pressure of the choroid epithelum cells. In other words, the cranio-spinal contents cannot any longer be regarded as a fixed quantity without the power of expanding or contracting in volume. An excess of carbon dioxid (or lack of oxygen) in the blood, volatile anesthetics, and a specific extract of the choroid glands or of the brain are capable of increasing the flow of the fluid. The fluid escapes from the cerebrospinal cavity by the lymph channels of nerves. A minor method of its escape is by the olfactory nerve, and clinical experience has shown that this route of escape is not a negligible one. The presence of the fluid within and around the structures of the central nervous system fulfills certain mechanical functions of support and pressure. The nervous mechanism of the body is so sensitive that the neurons must be bathed in an ideal physiological saline solution to maintain their osmotic equilibrium. Such a fluid is the cerebrospinal fluid. SACHS.

Puerperal Gangrene of the Extremities—A. STEIN, Surg., Gynecol., and Obstet., Oct., 1916.

The phenomena of incipient gangrene are identical with those of mechanical obstruction of the blood-vessels and naturally vary according to the mode of origin and the path of the infection in a given case. Pain is very pronounced and never absent in extensive vascular obliterations such as lead to peripheral gangrene. Infection usually being the etiological factor the ordinary symptoms of puerperal fever are apt to precede the signs of tissue necrosis. Septic puerperal endometritis is present in the majority of the cases. In milder cases only a rise in temperature will indicate some slight infection. The mode of onset of the gangrenous symptoms will sometimes, though rarely, suggest the cause and seat of the vascular obstruction. A sudden onset usually points to embolism in the arterial system (dry gangrene), while on the other hand the gangrene may develop very insidiously in the case of a small embolus. The onset of gangrene may be abrupt or gradual also in cases of purely venous obstruction (moist gangrene) so that the rapidity of evolution of the symptoms permits only an uncertain conclusion as to the seat and origin of the obstacle. In a general practical way it may be said that the early appearance of gangrene in the first few days of the puerperium points to an arterial (the most common) origin. The manifestations of puerperal peripheral gangrene do not require a lengthy description. Aside from the severe pain the absence of arterial pulsation below the thrombus or embolus is an important sign characteristic of the cases of arterial blocking. The sensibility is usually distinctly diminished, while the motility may be preserved. A livid discoloration and a relatively diminshed temperature of the affected area add to the probability of incipient gangrene. In cases of venous origin the inflamed veins of the pelvis are often distinctly palpable by the rectum or the vagina due to the existing thrombosis and periphlebitic edema. The two forms of dry and moist gangrene are apt to be combined in the same case, depending upon the rapidity of complete interruption of the blood current and the presence of external factors which favor mummification. Although the intolerable pain subsides as a rule when the gangrene has become established, the patient's general condition now becomes seriously aggravated through the absorption of necrotic tissue constituents. Before demarcation has occurred death may supervene, under increasing pulse and temperature, especially in cases of arterial obstruction. The prognosis of puerperal peripheral gangrene is governed in the first place by the timely performance of amputation. Unfortunately amputation is not always practicable on account of the bad general condition, the rapid progress of the gangrene, or because the seat of the obstruction is located high up in the aorta. Puerperal peripheral gangrene has accordingly a very bad prognosis as one-half to two-thirds of the patients die, for we are not able at the present state of our knowledge to prevent such an unfavorable outcome. While the mortality is still deplorably high, it has nevertheless been lessened by one-half since the institution of modern measures of treatment. The arterial, venous, or arteriovenous pathogeneses probably have no marked bearing upon the prognosis or the mortality although it appears that the mixed arteriovenous type of puerperal gangrene is credited with the most favorable figure. SACHS.

INFECTIOUS DISEASES

Etiology of Epidemic Cerebrospinal Meningitis—M. F. H. GAMBLE, Lancet, Aug. 12, 1916.

Author is convinced that epidemic cerebrospinal meningitis is due to a true gonorrheal inflammation of the arachnoid membrane of the brain and spinal cord. Such an infection he believes to be caused by the bites of gonococcal infected pediculi corporis.

SACHS.

Hemolytic Jaundice—C. SCOKALSKI, Zeitschr. f. klin. Medizin, Vol. LXXXIII. Nos. 3 and 4.

Description of a case of hemolytic jaundice concurring with hypertrophy of the liver and spleen, and leukopenia. Syphilis proved to be the etiological factor, as mercurial injections caused improvement.

WESTERN.

Weil's Disease—A. STOKES and J. A. RYLE, Brit. Med. Jour., Sept. 23, 1916.

About 15 cases of Weil's disease have come under observation by the authors and they have been able to confirm the findings of the discoverers of the pathogenic cause of the disease. The characteristic symptoms of the cases observed by them have been pain, which appears to be of a muscular character, in the head and lower limbs, vomiting, jaundice and extreme injection of the conjunctivae accompanied by great prostration. Experimental facts were meagre, but sufficient to show that the cause of these cases of Weil's disease (infectious jaundice or epidemic jaundice) is the spirochetosis icterohemorrhagica.

SACHS.

Bacillus Abortus—L. H. COOLIDGE, Jour. Med. Research, July, 1916.

By feeding a milk which contains the bacillus abortus (Bang) and which also contains the antibodies of this bacillus, it is possible to cause antibodies to appear in the blood of human adults. Nevertheless, the author states that he has no proof that this organism is pathogenic for human adults. He is of the opinion that the presence of such antibodies, so induced in the blood, indicates a passive immunity due to the absorption from the large intestine of the antibodies present in the infected milk.

SACHS.

The Gruber-Widal Reaction in Persons Vaccinated against Typhoid Fever—G. HERXHEIMER, Berliner klin. Wochenschr., July 24, 1916.

The Gruber-Widal reaction is hardly of any value in persons immunized against typhoid fever.

MILL.

Investigations concerning Typhus Fever—H. DA ROCHA-LIMA, Münchener med. Wochenschr., Sept. 26, 1916.

Despite many trials author never succeeded to infect lice in reconvalescents from typhus. This points to the fact that in typhus there are no healthy virus carriers. Even during and immediately after the decline of the fever the virus in the blood is so scarce that, as a rule, the lice cannot infect themselves with it any longer. It is sufficient that the louse sucks but once a febrile typhus patient in order to obtain the virus. The blood of typhus beginning with the fourth day of the disease gives rise to the Rickettsia infection in the louse. Guinea-pigs are of equal value as the lower monkeys as experimental animals. MILL.

A New Affection, Resembling Relapsing Fever—G. ZOLLENKOPF, Deutsche med. Wochenschr., Aug. 24, 1916.

Brief description of a new clinical picture. The affection is characterized by a sudden onset of fever, occurring every 4 or 5 days, accompanied by pain in the neck and stiffness, pains in the spleen and liver, pains in the articulations and the calves. By staining in accordance with May-Grünbaum some red cells present very fine dots and rods. It is possible that this new clinical picture has some connection with cerebrospinal meningitis. MILL.

Locally Limited Tetanus—H. F. BRUNZEL, Berliner klin. Wochenschr., Oct. 2, 1916.

The prognosis of a locally limited tetanus is not a priori a favorable one. It is best not to differentiate between a localized and a general tetanus. The local form, in case it remains really locally circumscribed, is only a special type of general tetanus. We know that general tetanus frequently sets in with local symptoms. MILL.

Reinfection in Lues—L. LICHTENSTEIN, Wiener klin. Wochenschr., Sept. 21, 1916.

History of a case in which a soldier reinfected himself by his wife who was formerly infected by him. The immunity after lues may already cease in the first year after the first infection so that a reinfection by means of the spirochetes which caused the original infection but retained their virulence may take place. The cessation of the immunity demonstrates that in this case the mercury-salvarsan treatment did not accomplish a cure in the bacteriologic-serologic respect. MILL.

Subfebrile Temperature in the Diagnosis of Incipient Pulmonary Tuberculosis—V. SCHLAEPFER, Zeitschr. f. klin. Medizin, Vol. LXXXIII, Nos. 3 and 4.

Of all diseases exhibiting subfebrile temperature, tuberculosis occupies the first place. The first examination of 41 per cent. of all cases revealing subfebrile temperatures also show clearly demonstrable tuberculous changes in the lungs. To this must be added 10 per cent. of the cases in which tuberculosis makes its appearance later on. Patients showing tuberculous symptoms in later years, frequently show in the beginning manifestations of neurasthenia, Basedow's disease and chlorosis. Patients, suffering with tuberculosis later on, furnish 25 per cent. of all patients with subfebrile temperature. Therefore 52 per cent. of all the patients with subfebrile temperatures are affected with manifest or latent pulmonary tuberculosis. Of the remaining 48 per cent. of cases with subfebrile temperature 32 per cent. show the symptoms of gastric ulcer and constipation, of neurasthenia, chlorosis and myalgia and 16 per cent. suffer with non-tuberculous, chronic inflammatory affections. The remainder of the article should be read in the original.

WESTERN.

Tuberculin Reaction in Scarlet Fever—R. LENNEBERG, Archiv f. Kinderheilkunde, Vol. LXV, Nos. 5 and 6.

Cutaneous and intracutaneous administration of tuberculin during scarlet fever showed that for the first few days the reaction-ability of the skin was diminished, but that the reaction-ability did not cease. Sensitizing of the body for tuberculin was frequently the consequence of the administration. MILL.

Complement Fixation in Tuberculosis—J. CORPER, Jour. Infectious Dis., Sept., 1916.

The complement fixation test for tuberculosis is positive in only about 30 per cent. of all clinically definite cases of tuberculosis, both active and inactive. When taken in conjunction with other findings a definite positive reaction makes the diagnosis of tuberculosis certain. A positive test was never obtained by the author in the absence of a positive von Pirquet reaction. SACHS.

Tuberculosis of the Cervical Lymphatics—C. M. DOWD, Jour. A. M. A., Aug. 12, 1916.

Many cases of enlarged cervical glands are treated as tuberculous which have not been so considered by the author. Many such nodes when removed will show only hyperplasia. Dr. Farr states that probably 5 patients present themselves in the Wilkes Dispensary with hyperplastic adenitis for every one with tuberculous adenitis. Many cases cited as operative failures are failures because of incomplete removal of the affected glands. It is possible for a patient to have a ruddy appearance and yet have masses of tuberculous lymph nodes in the neck. SACHS.

RESPIRATORY AND CIRCULATORY ORGANS

Cold Air and Blood Pressure in Pneumonia—J. L. MORSE and D. M. HASSMAN, Am. Jour. Dis. Child., Nov., 1916.

In children there is no constant relation between the systolic, the diastolic, or the pulse pressure and the severity of the pneumonia or the temperature of the surrounding air. The rates of both the pulse and the respiration show a tendency to vary directly with the temperature of the surrounding air. The patients symptomatically seem more comfortable when they are out of doors than when they are in the house. No conclusions are justified as to the effect of the cold-air treatment on the mortality of pneumonia in childhood.

SACHS.

Aneurism of Hepatic Artery—J. H. TEACHER and W. R. JACK, Glasgow Med. Jour., Nov., 1916.

Rowland, who has traced in the literature 40 cases of aneurism of the hepatic artery, states that in 24 cases the aneurism was extra-hepatic, in 8 intra-hepatic, and in 2 cases there was both an intra- and extra-hepatic aneurism. In some cases there have been no symptoms referable to the liver, rupture of the aneurism being the first indication that anything locally was wrong. In other cases the most common symptoms were pain, jaundice and hemorrhage. The hemorrhage occurs either from the stomach or intestine and a striking feature about it is that it occurs frequently, with varying intervals between the attacks. The pain usually resembles that of biliary colic. Jaundice is due to pressure on the hepatic ducts or on the common bile duct. The authors report the case of a man, 40 years old, apparently suffering from cardiac and renal disease who died suddenly from intra-peritoneal hemorrhage. The hemorrhage was found to come from the bursting of an aneurism in the left lobe of the liver. The condition was probably the result of a syphilitic infection. SACHS.

Bock's Stethoscope in Determining Myocardial Efficiency—J. RAMSAY, Brit. Med. Jour., Oct. 14, 1916.

Author states that in the Bock stethoscope we have an instrument which is helpful in estimating the functional efficiency of the heart muscle. When used in routine examination it may discover latent myocardial weakness. The principle of this stethoscope depends on the fact that the normal ratio of intensity of the first sound of the heart at the apex to the second sound at the base is approximately 2:1. The ratio is frequently altered when the myocardium is affected and it may become as 1:1. The sounds of the heart pass through a hole in the chest piece of the stethoscope to the observer's ear. The hole may be increased in size or entirely obliterated by turning a screw in one or the other direction. To the screw a pointer is fixed which works along a scale on which at equal intervals are the numbers 10, 20, etc., up to 100. When the point is at 100, the hole in the interior of the chest piece is entirely closed and no sound reaches the observer's ear. By moving firstly the pointer until the first sound of the heart is indistinguishable and then until the second sound is obliterated, one may obtain values from which the ratio of intensities of the sound may be computed.

SACHS.

Pericarditis—E. A. LATCH, Bost. Med. and Surg. Jour., Oct. 26, 1916.

In a study of the necropsy reports and the clinical records of the

Boston City Hospital for the past 19 years, 359 cases of true pericarditis were described in the records of 3683 sections. Out of the 359 cases of pericarditis, 150 were of the acute variety and in a study of the clinical records of these cases, only 17 per cent. gave any hint as to the existence of the disease. In these acute cases of pericarditis, rheumatism was the primary cause in two instances, pneumonia in 63, empyema in 6, tuberculosis in 7, local or general sepsis in 28, nephritis in 16, meningitis in 3, and in 17 cases the primary cause was not determined. SACHS.

Some Varieties of Congenital Heart Disease—L. FINDLAY and W. B. M. MARTIN, Glasgow Med. Jour., Oct., 1916.

Two cases of congenital heart disease are described in this paper, Case (1) A case of congenital stenosis of the pulmonary tract with atresia of the orifice—septal defects and patent ductus arteriosus. In this case of a child of 3 months, the mother noted that when the child was 2 weeks old, its body was of a bluish tint. Since that age it had a cough and was subject to "peculiar turns," in which the cyanosis became very marked and the breathing stopped. Examination of the heart did not disclose any enlargement of the precordial dulness and the heart sounds were pure. The pulse was 140-150 a minute. Autopsy revealed a congenital stenosis of the pulmonary tract with atresia of the orifice, incomplete interventricular septum, patent ductus arteriosus and patent foramen ovale. The primary factor in this case is a defective development of the pulmonary tract. The septal defects are inevitable consequences of the pulmonary atresia. The fact that the pulmonary orifice was completely occluded accounts for the absence of any murmur indicative of pulmonary stenosis. (Case II.) Congenital stenosis of pulmonary tract, with septal defects. This child was two months old. It was under observation in the hospital on account of a stationary weight. It was cyanosed and presented great distension of the veins over the chest and abdomen. The pulse was small and could not be satisfactorily counted. The precordial dulness was slightly enlarged to the right and all over the heart could be heard a blowing systolic murmur, with its seat of maximum intensity at the base. The second sound at both pulmonary and aortic areas was loud and sharp. While under observation the intensity of the cyanosis varied and at times the murmur could not be heard. Postmortem revealed an example of the commonest type of congenital heart disease, i. e., the combination of pulmonary stenosis with patency of the inter-ventricular and inter-auricular septa.

SACHS.

Clinical Significance of Sinus Arrhythmia—L. T. THORN, Practitioner (London), Sept., 1916.

Author states that sinus arrhythmia in young adults should not be disregarded, for it is not a normal condition and such individuals are more liable to suffer from a cardiac breakdown under mental or physical strain than those who have a perfectly regular pulse.

SACHS.

Third Heart Sound—E. W. BBIDGMAN, Heart (London), Vol. VI, No. 1.

The third heart sound was definitely audible in 13 of 16 normal boys, and was represented on the phonograms of all. It is accentuated by different maneuvres. Einthoven's calculations, whereby the third sound is regarded as being below the limits of audibility are questioned and comparisons are made which tend to show that the third sound is nearly of the same intensity as the first or second.

SACHS.

ALIMENTARY TRACT

Mucormycosis of the Stomach—O. TEUTSCHLÄNDER, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXIX, No. 2.

Report of a case and a discourse of the etiological possibilities of this rare condition. MILL.

Carcinoma of the Suprapapillary Duodenum—G. JEFFERSON, Brit. Jour. Surg., Oct., 1916.

Carcinoma of the duodenum is a rare disease. It is found in 0.04 per cent. of hospital post-mortems (i. e., deaths from all causes). Inch for inch, the duodenum is more liable to cancer than the rest of the small intestine. Of 71 small intestine carcinomata, 34 (48 per cent.) were in the duodenum. A casual relationship between cancer and simple ulcer is difficult to establish in the case of the duodenum. SACHS.

Bastedo's Sign in Chronic Appendicitis—J. ROSENBLOOM, Surg. Gynecol. and Obstet., Nov., 1916.

Bastedo's sign is produced by inflating the cecum and colon by means of a colon tube passed 10-12 inches into the rectum. If appendicitis be present, pain will appear at the site of McBurney's point when the colon is distended. Rosenbloom is of the opinion that this sign is of value in the diagnosis of chronic appendicitis.

SACHS.

Ineffective Appendicectomies—R. T. MORRIS, N. Y. Med. Jour., May 20, 1916.

In cases of syncongestive appendicitis we have a distension of the inner coats of the appendix with interstitial infiltrates, along with a similar infiltration of other organs besides the appendix, because of some organic disease, with obstruction to the blood and lymph circulatory systems. The reason why the appendix speaks up is probably because the inner soft coats cannot swell freely within the tight outer sheath of peritoneum, and an irritation of nerve filaments results in these appendices. It follows that in these two irritative lesions of the appendix, removal of that organ simply disposes of one unimportant factor in the patient's symptoms, and the cases as a whole must be taken into consideration or the operation will be a failure. A still larger group of cases in which the appendix is removed ineffectively includes patients who never had appendicitis at all. Concerning the acute cases author has commonly found that the symptoms were due to some infective lesion of the pelvis or some infective lesion of the stomach or gall-bladder, with accompanying tenderness of groups of abdominal sympathetic ganglia. Sometimes the cases have been bubonocele with pinching of a small area of bowel; sometimes they have been cases of pneumonia with pain referred to the right lower abdomen, and sometimes they have been cases of toxic irritation of spinal cord centres with efferent impulses sent out along the ilioinguinal and iliohypogastric nerves. The reason why these toxic neuroses appear oftenest on the right side is perhaps the fact that the ascending colon is such a fertile focus for enteric toxemia. Not only are patients of this group sent in frequently for operation at the hospital, but I am sometimes called to a distance and asked to be prepared for immediate operation upon the appendix. A still larger group of cases with symptoms in the appendix region, but not including the appendix, appears to depend upon a large and complicated variety of irritative lesions of the spinal cord, with efferent impulses sent out, not only to the cerebrospinal nerves, but also to the sympathetic nervous system, and possibly to the autonomic centres. SACHS.

Pseudo-Appendicitis—F. G. CONNELL, Jour. A. M. A., July 29, 1916.

The existence of such a clinical entity as chronic appendicitis has been questioned. The diagnosis is often incorrect and is being made with increased frequency. After eliminating all demonstrable pathological conditions that might possibly be confused with "chronic appendicitis," there remains in certain cases some cause for pain in the right iliac fossa other than the appendix, the exact cause of which is as yet not definitely known. Such cases of pseudo-appendicitis are frequently associated with viseral ptosis, constipation and neuresthenia. Pseudo-appendicitis is in no way related to the appendix and is a non-surgical condition in which appendectomy is contraindicated. The diagnosis of chronic appendicitis presupposes a previous attack of acute appendicitis. SACHS.

Accumulation of Pus or Bile beneath the Diaphragm as a Consequence of the Presence of Gall-Stones—RIEDEL, Deutsche med. Wochenschr., Aug. 31, 1916.

Pus or bile accumulations beneath the diaphragm as a consequence of biliary calculi ensue not frequently. They may occur as a result of the perforation of the fundus of a gall-bladder containing calculi when there exist adhesions with the anterior abdominal wall.

MILL.

The Activity of Physostigmin and Pilocarpin Combinations upon the Surviving Intestine—L. MOLDOWSKAJA, Zeitschr. f. experimentelle Pathologie u. Therapie, Vol. XVIII, No. 1.

Investigations concerning the tonic influence of physostigmin and pilocarpin on the longitudinal musculature of the intestine. The alkaloids exert a varying influence and have a pharmacologically different behavior throughout. The combination of both substances shows a greater total effect. This combination effect, however, ensues only when relative very small amounts of the one substance are added to relative large quantities of the other. Author demonstrated that it does not matter whether much physostigmin and little pilocarpin or much pilocarpin and little phystostigmin are employed. Combination of large quantities of both substances causes decline of the total activity. This decline appears to be in the nature of an inhibitory effect. WESTERN.

Bacteriology of Feces in Diarrhea of Infants—W. R. LOGAN, Lancet, Nov. 11, 1916.

The flora of the feces of artifically fed infants differs from that of breast fed infants chiefly in a decrease of the acid tolerant group, an increase of the normal B. coli group, and in the appearance of members of the non-lactose-fermenting group, along with some increase of cocci. The flora of infants suffering from diarrhea shows similar and more marked changes, and the more severe the diarrhea, the more marked the changes. It is doubtful whether the overgrowth of non-lactose-fermenting bacilli initiates the diarrhea or whether it is a secondary and aggravating factor. SACHS.

Observations on Dysentery—A. W. WAGNER, Lancet, Oct. 21, 1916.

Both in amebic and bacillary dysentery secondary invasion of the ulcerated intestinal wall by organisms from the intestinal lumen is an important factor aggravating both the local and general condition. The pyemia, so frequently observed in the late stages of amebic dysentery, is a result of this secondary invasion and though usually toxic in origin may be due to bacterial invasion of the blood stream. Amebic dysentery may be latent, the ulcers being confined to the cecum and producing no symptoms. Mannite fermenting dysentery bacilli can exist in the intestine in an avirulent form. The agglutination reaction in dysentery is a valuable means of differentiating the bacillary type of the disease. In Shiga infections specific agglutinins are invariably present, after the first week of the disease.

SACHS.

NERVOUS SYSTEM

Reducing Substances in Cerebrospinal Fluid—J. LÖWY, Zeitschr. f. klin. Medizin, Vol. LXXXIII, Nos. 3 and 4.

The sugar content of the blood in the same patient is always higher than in his lumbar fluid. In chronic affections of the central nervous system the amount of sugar fluctuates between 0.08 to 0.1 per cent. In inflammatory affections of the meninges and the central nervous system the reducing substances are not the exclusive expression of a transudation process from the blood. A part of the sugar is probably caused by inflammatory processes in the central nervous system. When the prevailing hyperglycemia is marked the sugar content of the lumbar fluid is also increased. Acetone and diacetic acid are demonstrable in the cerebrospinal fluid. A glycolytic process does not seem to take place in the cerebrospinal fluid. WESTERN.

Chvostek's Sign—A. FUCHS, Wiener klin. Wochenschr., Sept. 7, 1916.

Further observations on the analogy and identity of the symptoms of tetany and those of ergotism. Author has succeeded in removing Chvostek's sign in patients not affected with tetany by removal of the flour residue from the intestine and the non-ingestion of flour. It is very probable that Chvostek's sign does not stand in pathogenic relationship or symptomatologic connection with any disease, but represents a region of increased irritability of the central nervous system which is caused by a poison generated by ergot and which shows itself normally in the form of a tonic activity, but which appears pathologically as overirritation (spasm). MILL.

Chorea—I. A. ABT and A. LEVINSON, Jour. A. M. A., Nov. 4, 1916.

Authors believe in the relationship between chorea and rheumatism. Tonsillitis was not a predominant factor in the authors' series of cases. The mortality in this series of cases was one per cent. Arsenic does not seem to have any effect on the duration of the disease. SACHS.

Epilepsy in Offspring of Epileptics—D. A. THORN, Bost. Med. and Surg. Jour., Oct. 26, 1916.

Author is of the opinion that epilepsy is less frequently transmitted directly from the parent to the off-spring than has heretofore been believed. This conclusion is based on the study of 33 cases.

SACHS.

Bacillus Isolated from Epileptics—W. B. TERHUNE, Jour. A. M. A., Oct. 14, 1916.

Author states that although he began his investigations profoundly biased, as he believed that epilepsy was not bacterial in origin, he is forced to the conclusion that the bacillus which he has isolated and which is identical with the bacillus epilepticus described by Reed, must be an etiological factor in epilepsy. The bacillus was isolated in 75 per cent. of epileptics examined. It was present during and following a seizure but not during the intraconvulsive period, except in the case of one patient who was debilitated by ill health. It was not found in non-epileptics. It causes typical epileptoid convulsions in cats during which death may occur, both when they are injected intravenously and when they are fed cultures of the organism. The organism may be recovered from the animal during the convulsion and after death. SACHS.

Disturbance of Vision from Cerebral Lesions—G. HOLMES and W. T. LISTER, Brain (London), Vol. XXXIX, Parts I-II.

The authors state the following conclusions which cannot be regarded as final: That the upper half of each retina is represented in the dorsal, and the lower half in the ventral part of each visual area; the center for macula or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital poles; that portion of each upper quadrant of the retina in the immediate neighborhood of, and including the adjacent part of the fovea centralis is represented in the upper and posterior part of the visual area in the hemisphere of the same side, and vice-versa. The center of vision subserved by the periphery of the retina is probably situated in the anterior end of the visual area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forwards in the visual area. SACHS.

A New Familial Infantile Form of Diffuse Brain-Sclerosis—K. KRABBE, Brain (London), Vol. XXXIX, Parts I-II.

The so-called diffuse sclerosis of the brain in children may be divided into at least three quite distinct types: (1) A syphilitic form; (2) Schilder's encephalitis periaxialis diffusa; (3) a familial infantile form of which 5 cases are discussed in the present paper; the literature probably contains one other case. This form shows the following characteristics: It is usually a familial disease; it sets in somewhat acutely in about the fifth month in a child who up to then has been quite healthy; it progresses on a chronic course, ending with death, five to six months after the onset; universal rigidity of the musculature, violent tonic spasms, probably causing pain, and brought on by touching or noise form the characteristic symptoms. As a rule nystagmus is present, and in the later stage, atrophy of the optic nerve. Periodic elevations of the temperature occur without perceptible cause, outside of the central nervous system. Finally extensive paresis and pronounced debility close the scene. The pathologico-anatomical findings are: a marked hardness of the white substance of the brain without alteration of its shape. Complete destruction of the white matter of the cerebellum and degeneration of the spinal nerve tracts are present. The affection must be regarded as a purely degenerative and not as an inflammatory process. SACHS.

URINARY ORGANS—MALE GENITALIA

Termination of Acute Nephritis—B. GOLDBERG, Deutsche med. Wochenschr., Aug. 24, 1916.

After the disappearance of the albumin in the urine, the last and most stubborn manifestation of acute nephritis is the presence of red cells in the clear yellow urine. These erythrocytes persist for from 5 to 9 months. Their disappearance indicates the complete recovery of the patient. MILL.

Acute Nephritis in War Participants—BRUNS, Zeitschr. f. klin. Medizin, Vol. LXXXIII, Nos. 3 and 4.

Nephritis of war participants frequently concurs with edematous swellings which need not necessarily be of renal origin. The urine contains albumin and much blood. The presence of the blood may persist for a long time. Uremic manifestations are of frequent occurrence. Cases of uremia with eclampsia have no retention-nitrogen increase in the blood. The blood pressure is increased during the period of edema, declining with the disappearance of the latter. Almost 50 per cent. of the patients exhibit during the stage of edema, a rention-nitrogen increase in the blood. The subacute stage of these nephritides persists often for months, until, finally, the last traces of albumin and red blood cells disappear. This form of nephritis is prone to recur frequently. The recurrent nephritis is clinically the absolute repetition of the original affection. Chronic nephritis seems not to result from an acute attack. Generally speaking, therefore, the affection bears a benign character. WESTERN.

Hemorrhagic Nephritis in War Participants—H. ZONDEK, Zeitschr. f. klin. Medizin, Vol. LXXXIII, Nos. 3 and 4.

Nephritis in war participants offers in functional respects no essential characteristics differing from the manifestations of the disease in non-participants. It is a form of the azotemic-nitrogen-retention type. Sodium chlorid metabolism appears not to be retarded. On the contrary, the kidneys excrete much NaCl and this increased NaCl excretion may last for weeks. This special form of nephritis may functionally be amenable at the time when the urine still contains about one-half pro mille of albumin, some casts and a moderate number of red cells. An extreme limitation of NaCl ingestion is not essential for therapeutic purposes. WESTERN.

Pyonephrosis—B. KOWALSKI, Monatsschr. f. Geburtshilfe u. Gynäkologie, Vol XXXXIII. No. 5.

Three factors must be taken into due account when confronted with the pathologic-anatomical picture of pyonephrosis, (1) the mode of infection (ascending or descending); (2) the type and virulence of the pathogenic microorganisms, and (3) the duration of the affection. MILL.

Non-tuberculous Renal Infections—H. CABOT and E. G. CRABTREE, Surg. Gynecol. and Obstet., Nov., 1916.

Authors state that pyogenic and non-pyogenic organisms produce essentially different kidney lesions. Organisms producing renal infections appear in the urine. The organisms of pyogenic infections may appear only during the early stages and as a rule there will be little or no amount of pus for they are shut off from the lower portion of the kidney and do not involve the membrane of the pelvis. The non-pyogenic organisms are chiefly formed in the kidney pelvis and there produce a considerable amount of pus. There is little disturbance of renal function, as is evidenced by the phthalein test, when the kidney is infected by pyogenic organisms but a colon bacillus infection markedly influences the renal function for this organism produces lesions firstly on the convoluted tubules and then on the renal pelvis. SACHS.

FEMALE ORGANS OF GENERATION — PREGNANCY — PARTURITION—INFANTS

Fertility and Sterility—E. REYNOLDS, Jour. A. M. A., Oct. 21, 1916.

The reactions of the spermatozoa to the vaginal and cervical secretions are quite different and must be considered separately. The vaginal secretion is normally acid, bacteria-laden and varyingly hostile to the spermatozoa even when admixed with the alkaline semen. The hostility of the vaginal secretions to the spermatozoa is the result of the bacterial conditions which belong with the highly acid secretions. The cervical secretion is normally bacteria free and therefore physiologically favorable to the spermatozoa. It is, however, often mechanically hostile to them. Cervical mucus enmeshes and renders motionless many spermatozoa.

SACHS.

Wassermann Test in Pregnancy—F. H. FORBES and J. J. MOORE, Jour. A. M. A., Aug. 19, 1916.

Syphilis is usually a latent condition in pregnant women. The majority of mothers having syphilis are ignorant of their condition and therefore improperly treated. In a series of 160 pregnant women 11.3 per cent. gave a positive Wassermann. In 116 married women, 10.6 per cent. gave a positive reaction. In 44 single women 13.5 per cent. gave a positive reaction. Only one of 18 giving a positive reaction had a history of syphilis, 6 gave histories of previous abortions, 3 had severe complications of pregnancy, as eclampsia and mental psychoses. SACHS.

Vicarious (Complementary) Menstruation—PULVERMACHER, Zentralblatt f. Gynäkologie, No. 35, 1916.

Report of 3 cases of vicarious menstruation. In one case the hemorrhage took place from a fecal fistula which resulted from a perityphlitic abscess. It ensued regularly every four weeks and lasted from two to three days. In the other two cases the hemorrhage ensued through one of the nipples. MILL.

Menstruation-Resembling Hemorrhages during Pregnancy—J. POK, Gynäkologische Rundschau, Vol. X, Nos. 3 and 4.

Menstruation-like hemorrhages may regularly appear during pregnancy and may be mistaken for a menstrual process. During the first 3 or 4 months, these pseudomenstruations resemble a genuine menstruation, but the bleedings become less as the gestation period progresses. They bring about an absence of the subjective pregnancy symptoms until the beginning of the fetal movements. A more pronounced pseudomenstrual hemorrhage may induce abortion or premature birth. MILL.

Retraction of Uterine Muscle and Obstructed Labor—H. T. HICKS, Brit. Med. Jour., Oct. 14, 1916.

Hicks states that a retraction of the uterine muscle producing obstruction of labor is not often recognized. In such cases, the fetal head does not engage in the pelvic brim for the latter is usually contracted or distorted. The membranes rupture early, the cervix is unfilled and later on the uterine muscle passes into a state of tetany. The uterine muscle becomes hour-glass in shape with the neck of the fetus in the contracted portion. Retraction progresses, the upper segment moulding itself to conform to the shape of the fetal body while he lower segment becomes progressively thinner. A high position of the head in a primipara, a short woman, with a square head and short long bones, should at once put one on their guard for the possibility of the existence of this condition. SACHS.

The Tubes in Puerperal Fever—GASSOT, Korrespondenzblatt f. Schweizer Aerzte, 1916, No. 34.

Author found pyosalpinx in but one instance of 9 necropsies of patients who had died from puerperal infection. This pyosalpinx originated secondarily. In another instance he found an old tubal catarrh. He concludes that the tubes play no important part in the spreading of the infection. Besides, pyosalpinx ensues but rarely on the basis of a puerperal infection; it is due in most every instance to a gonorrheal invasion. MILL.

Primary Carcinoma of the Vulva—A. STEIN, Am. Jour. Obstet. and Dis. of Women and Child., Vol. LXXIV, No. 4, 1916.

Carcinoma of the vulva may not cause any disturbances for a considerable length of time and is apparently painless until the growth has ruptured through the skin. These patients are, therefore, not apt to seek advice before ulceration has begun and often complain only of local soreness and a burning sensation on micturition. There is usually a history of a small painless nodule or a superficial ulcer with a tendency to bleed more or less, gradually increasing in size without serious disturbances of a local or general character. Profuse hemorrhage and discharges are rare and never appear until late in the disease. Pruritus is in many cases one of the earliest symptoms of vulvar carcinoma. On examination the location of the tumor in the vulva in the majority of the cases is discovered in the labia majora, more particularly on the internal surface. Swelling of the lymph glands in the groin is almost invariably demonstrable no matter how small and apparently insignificant the vulvar tumor. This premature enlargement of the inguinal lymphatics is probably not the result of infection of the tumor surface associated with inflammatory swelling, but in view of the very frequent glandular recurrences after radical operations must be interpreted as a manifestation of early metastasis. The malignancy of carcinoma of the vulva as well as the symptomatology are illustrated by the following instructive case, recently reported by Frigyesi in Budapest. The patient, a woman 46 years of age, noticed one year ago a swelling of the vulva the size of a hazel nut, which caused severe itching and was removed. When she came under observation 10 months later, the vulva on examination was found to be reddened, swollen and painful; the inguinal lymph glands on both sides were more or less enlarged up to the size of a bean. The right labium majus was shrunken and atrophied, whereas the left labium majus and minus were occupied from the prepuce of the clitoris downward by a large cartilaginous tumor the size of a fist, with an eroded and bleeding inner surface. The growth began in the middle line extending anteriorly to the urethral bulb and posteriorly for a distance of

1 1-2 cm. to the vaginal wall. Exploratory excision showed the tumor to be a carcinoma. The recognition of cancer of the vulva is usually easy. Syphilis and tuberculosis having been excluded, the growth can in most cases be identified without difficulty by the induration which extends deeply into the connective tissue; the cauliflower-like smeary surface; and the crater-like eroded margins. An exploratory excision of tumor tissue is very rarely required. The microscopical findings in these cases do not differ in any particular from the cutaneous carcinomata affecting other regions of the body.

SACHS.

Renal Secretion in the Nursling—G. TUGENDREICH, Archiv f. Kinderheilkunde, Vol. LXV, Nos. 5 and 6.

Observations in a nursling with ectopic bladder. The right kidney excretes more urine than the left; during sleep the urinary secretion is diminished; more urine is secreted during the day than at night.

MILL.

Technic for Intravenous Injections in Infants—A. MARTIN, Brit. Med. Jour., July 8, 1916.

On account of the size of the superficial veins in young infants it is usually difficult to give intravenous injections or even to obtain blood for the Wassermann reaction. This difficulty may be overcome by puncturing the longitudinal sinus through the anterior fontanelle according to the procedure recently introduced by Helmholtz, of Chicago. This method is free from risk. The scalp over the anterior fontanelle is shaved and disinfected with iodin. The child lies in bed with the head raised on a pillow. The needle is held at an angle of about 50-60 degrees with the plane of the fontanelle and is inserted in the middle half way, between the anterior angle and the center of the fontanelle. Some little force is required to force the needle through the firm pericranium and it should not penetrate more than an eighth of an inch below the surface of the skin since the sinus lies immediately below the pericranium. If the needle is in the sinus, blood flows at once into the syringe. Should this not happen, the needle is too deep, but no harm has been done. A needle of size No. 20 with a short bevel measuring 2 mm. should be employed.

SACHS.

Vaginitis in Infants—A. F. HESS, Am. Jour. Dis. Child., Nov., 1916.

Post-mortem examinations show that in the acute and subacute cases of vaginitis in infants, the cervix is most frequently involved and that the vagina generally shows no signs of inflammation. Cervicitis would therefore seem to be a more correct term in this condition than vaginitis. Where numerous pus cells without bacteria are found in smears made from the cervix, an inflammation may be assumed to be present, and in the overwhelming majority of instances the inciting factor will be found to be the gonococcus. Other microorganisms may, however, be the cause of the inflammatory process. It should be borne in mind that smears taken from new-born infants very frequently show pus cells, probably due to the invasion of the vagina by saprophytic bacteria. In the new-born infants, such a condition should not be considered pathologic. SACHS.

Gynecological Urology—H. D. Furniss, Providence Med. Jour., March, 1916.

The ureter catheter is of great value, but is too often used. The best çystoscopist is the one who has to use it the least. When the amount of pus coming from a kidney is small, or when we wish to determine the absence of disease of one kidney if the other is diseased, it is of great value. By its use we can detect obstructions, and when this obstruction is due to a calculus not shown by the X-Ray, its presence can be made known by the scratches made on a catheter waxed and passed by the stone. The location of shadows suggestive of stone can be determined by making other radiographs after introducing a catheter impervious to the Röntgen rays. A large percentage of women complaining of urinary symptoms have chronic urethritis and with the usual examination these are missed. For these I prefer the Kelly endoscope with the patient in the knee chest posture or the Gerringer endoscope with the patient on the back and the bladder and the urethra distended with water. In uretro-vaginal fistula we may have one or both ureters involved and the ureter may be so injured that all of the urine escapes into the vagina, or a part into that vagina and the rest into the bladder. In other words, a complete solution of continuity of the ureter or an injury involving only a portion of the tube. There are other finer points concerning ureteral fistula that space prevents considering. In diagnosing uretero-vaginal fistula we proceed as follows: Ten s.s. of a 3 per cent. solution of indigo-carmine is injected intravenously, the bladder filled with a boric acid solution. The elimination of the dye is observed through the cystoscope. Failure to see it coming from one side, and finding it in the vagina clinches the diagnosis of uretero-vaginal fistula on the side of the non-appearance. Appearance from both ureters and appearance in the vagina of the deeply stained urine speaks for an incomplete uretero-vaginal fistula. The passage of a ureteral catheter may be obstructed at the point of injury, but not necessarily so.

The Normal Baby—U. S. Public Health Service, Supplement No. 10.

An average healthy baby weighs from 7 to 7 1-2 pounds at birth; 15 pounds at 5 or 6 months, and 21 pounds at 12 mouths. In other words, the baby doubles its weight in 6 months and trebles it in

12 months. It is 20 to 21 inches long at birth, 25 to 26 inches at 6 months, and 28 or 29 inches at 12 months. More rapid gains are noted in the first 6 months than in the second 6 months. The average weekly gain is about 4 ounces. It sleeps soundly. Is happy, active, and enjoys using its arms and legs freely. Begins to follow moving objects with its eyes at the second or third month. Begins to sit unsupported at the seventh or eighth month. Cuts its first tooth from the sixth to ninth month; has about 6 teeth at 12 months, 12 teeth at 18 months, 16 teeth at 24 months, and 20 teeth at 30 months. Walks from the fourteenth to the seventeenth month. The soft spot or opening in the skull closes between the eighteenth and twenty-fourth month. Begins so say words like "papa" and "mamma" after the twelfth month, and simple, short sentences at the close of the second year. Children, however, that are otherwise perfectly normal may not begin to speak until a year later than the time stated above, or may present variations from any of the above.

SACHS.

THE

ARCHIVES OF DIAGNOSIS

Editor, Heinrich Stern, M.D., LL.D.
250 West 73d Street, New York, N. Y.
To whom all Communications of an *Editorial* Nature should be addressed

Publishers: Rebman Company
141, 143 and 145 West 36th St., New York, N. Y.

PRICE OF SUBSCRIPTION PER YEAR
(payable in advance)

To Subscribers in the United States, Canada and Mexico........... $2.00
To Subscribers in Other Countries (Postage prepaid)............... $2.50
Price of Single Copies, 75 Cents
The volume consisting of four issues comprises at least 400 pages

NOTICE TO CONTRIBUTORS

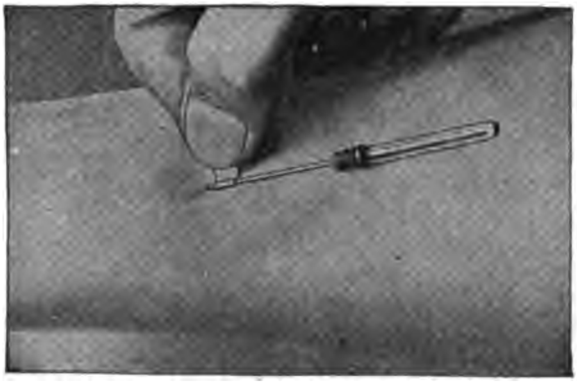
1. *The purport of the Archives is to publish papers which embody the results of original work and observation pertaining to Diagnosis and Prognosis.*
2. *Papers treating on Early Diagnosis, Functional Diagnosis, Therapeutic Diagnosis and Differential Diagnosis are especially solicited.*
3. *Contributions must not be of unreasonable length.*
4. *No contribution that has already appeared in print will be accepted for publication.*
5. *Galley Proof and Page Proof will in all instances be sent to the author for correction.*
6. *Contributors will receive 200 copies of their papers in pamphlet form free of charge. Additional copies will be furnished at cost.*

The Luetin Intradermic Test

In the Mulford Special Intradermic Test Syringe
A Simple and Accurate Method of Diagnosing Syphilis

Luetin is an extract of killed cultures of a number of strains of the Treponema pallidum (Spirocheta pallida) carefully sterilized and placed in sterile glass ampuls or intradermic syringes.

A positive reaction manifests itself in the form of a pustule, papule, or other inflammation at the site of injection.



Illustrating the Mulford Intradermic Syringe and Method of Testing. Hold needle by finger rest and carefully insert needle between the layers of the skin previously sterilized. Push intradermic needle up into the capillary tube and the proper amount of luetin is injected intradermically.

The Luetin reaction is specific for syphilis.

It occurs most constantly and intensely during tertiary and latent stages.

It is usually absent, or very mild, in primary or secondary stages, although in these stages it may become positive after energetic treatment.

In infants it is less marked than in adults with congenital syphilis.

The Luetin Test is furnished

In single test intradermic syringes.
In five-test intradermic syringes.
In ampuls containing 50 tests (without syringes) for hospital use.

Working Bulletin on Mercurialized Serum and Luetin Mailed Upon Request

---

H. K. MULFORD CO., Philadelphia, U. S. A.

Manufacturing and Biological Chemists

New York
Chicago
St. Louis
Atlanta
New Orleans
Minneapolis
Kansas City
San Francisco
Seattle
Toronto

London: 119 High Holborn

REBMAN COMPANY

141 W. 36th St. NEW YORK

A special Diploma and Bronze Medal have been awarded under section "Arts and Sciences" to Rebman Company, of New York, for an exhibit of their Books, by the International Jury of Awards of the Panama-Pacific International Exposition held in San Francisco in 1915.

A TEXTBOOK

OF

NERVOUS DISEASES

FOR STUDENTS AND PRACTISING PHYSICIANS

IN THIRTY LECTURES

BY

ROBERT BING

Dozent for Neurology at the University of Basel

ONLY AUTHORIZED TRANSLATION

BY

CHARLES L. ALLEN, M.D., Los Angeles, Calif.

WITH ONE HUNDRED AND ELEVEN ILLUSTRATIONS IN THE TEXT

Of especial value just now on account of Lecture XVI, which deals entirely with Infantile Paralysis

"The book has intrinsic value and will be found a real help in many perplexing and anxious moments in the physician's busy life. The illustrations are also very good and practical. Perhaps one of the more valuable features of the book is the "index" at the end of the volume. The author states at the beginning of his book that he lays great stress on a complete index, and he has given a very comprehensive general index and a very valuable index of authors."—*Woman's Medical Journal.*

"The illustrations are excellent and far better than what is found in most textbooks on nervous diseases. Altogether, it is one of the best and most satisfactory works on nervous diseases that has appeared for some time, and is far better than some of the more recent books by American authors. From the point of view of the student, it is in the reviewer's mind the best work that has appeared for many years."—*Amer. Journal of Medical Sciences.*

Cloth, $5.00

Address: REBMAN COMPANY

141 West 36th Street NEW YORK CITY

BURR PRINTING HOUSE, NEW YOR